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An Address.[1]

SIR MORELL MACKENZIE: A MEDICAL VISIONARY.

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IN this short presidential address to the Section of Oto-Rhino-Laryngology on the occasion of the fifth session of the Australasian Medical Congress (British Medical Association) it seems fitting that the centenary of the birth of Sir Morell Mackenzie should be recognized. As the fair city of Adelaide looks back proudly to the centenary of its birth and to its subsequent achievements, so should we, as a body of oto-rhino-laryngologists, carry our minds back a century to July, 1837, the year in which was born Sir Morell Mackenzie, one of the greatest pioneers in our branch of surgery, a protagonist of specialism and a strong character in whose steps we are proud to follow.

Born at Leytonstone, Essex, he came of medical stock, his father and uncle being well-known practitioners. His brother, Dr. Stephen Mackenzie, likewise carried on the tradition after him. After a distinguished career as a student at the London Hospital he was admitted a member of the Royal College of Surgeons in 1858. His post-graduate sojourn in the various countries in Europe marked an epoch in his life, during which his friendship with Czermak at Pesth developed. The singing

[1] President's address, read before the Section of Oto-Rhino-Laryngology at a meeting of the fifth session of the Australasian Medical Congress (British Medical Association) in August, 1937.

master, Garcia, had invented the laryngoscope which Czermak the physiologist was then using. Quick to seize on its possibilities in the laryngological sphere, Mackenzie adopted it in his armamentarium and eventually he was one of the foremost to popularize its use in Britain.

From a resident post at the London Hospital he ascended to the position of medical registrar, and subsequently he became assistant physician in 1866, and later full physician on that staff. For several years during this period he was lecturer in physiology jointly with Dr. Hughlings Jackson. But his forte was already laryngology, for in 1863 he was awarded the Jacksonian Prize for his essay "On the Pathology and Treatment of Diseases of the Larynx", and in the same year he opened a dispensary for diseases of the throat, now known as the Throat Hospital, Golden Square. Many of its house surgeons and post-graduate students no doubt are attending this congress today.

He also took an active part in forming the various special departments at the London Hospital, and his enthusiasm for specialism as a means of advance in the study and treatment of disease is shown in his writings, which excited a good deal of attention.

His success from his commencement in practice in 1862—just seventy-five years ago—was remarkable; yet the long list of his contributions to medical literature indicates the untiring energy of this medical Hercules. He was a corresponding member of the medical societies of Vienna, Pesth and Prague, and an honorary Fellow of the American Laryngological Association; he was president of the Section of Laryngology at the International Medical Congress at Copenhagen in 1884 and first president of the Laryngological Society in 1888. Small wonder that in the year 1887 he was called to attend the late Emperor Frederick, this being the most critical episode in Mackenzie's professional life; and it was in recognition of his services on that occasion that Queen Victoria, in 1887, bestowed on him the honour of knighthood. The Emperor also conferred on him the Grand Cross and Star of the Hohenzollern Order.

But Sir Morell's prominence was due to more than technical ability. He undoubtedly possessed great literary powers based on a liberal education and wide reading. Of an impressive and attractive manner, he was also a capable speaker and a dogged fighter when the occasion arose, as it did on various occasions during his illustrious career. And on such occasions with much foresight he was apt to apply the French maxim *"Toujours l'audace"*, and, suiting the action to the word, to "carry the war into the enemy's territory". Thus in 1869 "the battle of the special hospitals" was in the hands of a competent general as far as Sir Morell's specialty was concerned. The beginning of Britain's largest special hospital for diseases of the ear, nose and throat is of interest.

Soon after commencing practice in 1862 Mackenzie chose two rooms in Golden Square, London, as a suitable site for a clinic, and put up a placard reading thus: "Metropolitan Free Dispensary for Diseases of the Throat and Loss of Voice." There had never before been any special throat dispensary, and to this the patients flocked. By the end of the second year 5,915 patients had received treatment therein and its success excited adverse criticism from a section of the profession; "but the patients who took the physic did not as a rule read the criticism, and so no great harm was done".

The year 1878, according to Haweis, is memorable as deciding whether or no it would be possible at once to deal an effective blow at specialism and thereby damage the professional career of Mackenzie by an attack upon the Throat Hospital, Golden Square. Up to a certain point this succeeded, in that the Prince of Wales and the Duke of Cambridge resigned as office-bearers and the Hospital Sunday Grant was withdrawn. Morell Mackenzie was not at all beaten, however; with characteristic vigour and foresight he consolidated his forces at a subscribers' meeting and refuted the charges *seriatim* in an able and convincing speech. The treasurer of the Sunday Fund resigned and a modification of procedure took place. In 1891 the attendances at the hospital rose to 37,319 for the year. In 1936 the hospital had 100 beds and an annual out-patient attendance of over 60,000.

Mackenzie's average day was stated to be fourteen hours of steady professional toil, after which he sat on into the night working on the proofs of the long series of articles and the books which went into circulation. There is no intention in this short address of enumerating his many contributions to medical literature, of which his work "Diseases of the Throat and Nose" is outstanding. When writing in lighter vein he possessed the unusual faculty of combining instruction with amusement. In his essay on smoking he counsels moderation in the use of the "narcotic weed". He proceeds:

> I am not a member of the Anti-Tobacco League, nor do I believe that all those who seek solace from the herb nicotine:
>
> Go mad and beat their wives;
> Plunge, after shocking lives,
> Razors and carving knives
> Into their gizzards.

Haweis reflects that his grace and facility with the pen are apt to conceal from the reader the solidity of his literary performance. In advocating specialism in *The Fortnightly Review* in June, 1885, he states that a scientific man nowadays, who like Bacon "take all learning to be his province", would be

> in danger of being sent to associate with kindred enthusiasts in Bedlam. Such leviathans of omniscence loom dim and gigantic through the vista of the past like the megatherium and mastodon of remote geological periods, and the type is utterly extinct. In fact, the *Zeitgeist* looks with suspicion on universal learning and inclines to believe that the soundness of a man's knowledge is in the inverse ratio to its extent.

In more modern times Americans tersely describe such "leviathans" as "general specialists".

Mackenzie's use of a lay journal at that time suggests that he was dissatisfied with the regular medical publications, and that, as "all is fair in love and war", he felt himself pressed to circumvent the enemy existent within the profession. Written fifty-two years ago, the article should be read by us all in full as a fair and strong exposition of the subject. With an erudition seldom found nowadays in our profession, he called to his aid a knowledge ranging from philosophy to the arts. Such was his vision of the future and of the advance of specialism that he forecasted our present position in the community as specialists and wondered

> whether we shall ever advance so far in the subdivision of labour as to have no doctors at all, but such as are specialists, or whether such a state of things would be desirable.

Yet in a recent issue of *The Pharmaceutical Journal* in New South Wales it is noted that in Budapest in September, 1936, there were 2,697 registered medical practitioners, which is equal to one doctor in every 370 persons. Only one-fifth of those in practice are general practitioners: the four-fifths are specialists. And so we are reminded of Mackenzie's question, is such a state of things desirable? Better that a Plautus in our ranks preach moderation in our numbers and that we consolidate our gains as a whole and think rather in terms of quality than of quantity.

Public demand, however, and increasing pressure in the ranks of the general practitioner have created a greater number of specialists, thereby evolving a machine which threatens to make practice economically unsound and would now appear to be beyond our control. In New South Wales ear, nose and throat specialists quadrupled in number during the post-war years and joined far and wide in the scramble for honorary appointments. Earned incomes were correspondingly lower, and in the endeavour to increase their clientele and survive these specialists began to take multiple hospital appointments. Men's lives are now spent in the chores of honorary service at public hospitals instead of in private practice or in laboratories. Less time is spent in clinics abroad and original lines of thought are less frequently pursued. The machine has evolved a profession top-heavy with specialists.

However, Mackenzie's fight was within the profession for the establishment and recognition of specialism. In recent years it has become a fight by an increasing army of specialists against economic circumstances. Small wonder that the nationalization of medicine creeps ever nearer. What in Mackenzie's day was gladly given to the poor in public hospitals is now being dispensed to such an increasing proportion of the population that not only should the Government be given the facts, but, if Mackenzie's practice was followed, the general public also would be educated to the true position through the medium of the lay journals and other avenues of publicity. Specialists are no longer so willing to shoulder in public hospitals a responsibility that is clearly one for the whole community; and on this point Mackenzie, charitable as he was, would have been adamant.

In a recent address before the Royal Australasian College of Surgeons the following remarks were made: "The minor specialists have increased rapidly . . . Is the day of the general surgeon passing? I think the writing is on the wall for those who care to read." Considered with Mackenzie's statement that "the hostility to specialism not only originated within the medical profession, but has been all along almost entirely confined to that body", the modern attitude emanating from such an august college affords a sharp contrast to that of half a century ago.

Mackenzie believed, moreover, that

> the hatred of specialism was suppressed rather than extinct. The change, such as it was, was entirely due to pressure from without. Public opinion had in fact declared itself with such emphasis on the side of the specialist that the profession had been coerced into sullen acquiescence in the inevitable.

Meanwhile the sturdy growth of the "minor specialist" (*sic*) of last century has transformed him into the "major surgical specialist" of today. Special technique has been evolved and special instruments, for example, the bronchoscope, requiring long and frequent practice for their complete mastery, have made the specialist indispensable.

But has oto-laryngology progressed as far in Australia as it has done abroad? In the case of the neck, for instance, is there not a tendency to recede from the external neck surgery which is routine work in the throat clinics abroad? In many clinics in Australia external neck surgery is still regarded as the general surgeon's demesne. While intrapharyngeal surgery is permitted in a throat clinic, the excision of cervical glands by the laryngologist is regarded unfavourably. A youthful surgeon once seriously informed me that the laryngologist's work was to examine, diagnose and report on a malignant larynx, but the removal thereof was his task.

You all know that this is not the practice in Europe and America. What are the reasons for such retrogression on our part, if any? They are, I think, two: (i) our early technical training, both in anatomy and in external neck surgery, may be deficient; (ii) our younger men, even if trained abroad in such work, return to Australian clinics in which this class of work may be diverted to general surgical departments. Thus, starved of material, the hand soon loses its cunning. The remedy lies with ourselves: (i) the young surgeons should spend a longer apprenticeship in general surgery before specializing; (ii) most Continental throat clinics present better opportunities for the development of technique in external neck surgery, and more *post mortem* material is available there than in throat clinics in Britain or Scotland; (iii) the establishment of throat hospitals would allow us to control work of this nature; (iv) patients requiring laryngectomy should be given to a very few laryngologists, whose technique would thereby be perfected. This procedure is followed in Sweden.

More is necessary, however, than the mere recognition of technical ability in specialists. To what extent has recognition of our status been granted in our medical councils and academic circles? Chairs in special subjects, for example, in oto-rhino-laryngology, have been established for many years in foreign universities. While lack of population and of finance may still justify tardiness in their establishment in Australasia, it may justifiably be asked whence comes the opposition in Great Britain, where some universities are well endowed. Is it from within the profession, as it was seventy-five years ago? Meanwhile, lectureships and clinical teaching in special subjects have been established in our medical schools. It is noted with satisfaction that the British Medical Association and the Royal Australasian College of Surgeons established special sections within these organizations, and it remains with members of these special sections to prove themselves worthy, as they have in the past, of election to the governing councils of these bodies.

Were Mackenzie to pass judgement on the advance made since his day he would no doubt feel satisfied that the torch had been worthily borne in a brief half century. Were he content that his specialty had been raised to a sufficiently dignified position among allied sciences he would not so much stress what it had gained as ask what it had given to the common good.

On this there can be no argument. The scientific publications since his time tell the story, and it may be claimed fairly that this specialty has contributed its quota to research. Take, for example, the single subject of diseases of the throat. Writing in 1885, Mackenzie declared:

> The scientific literature relating to these dates from little more than twenty-five years back, and already it has grown to a bulk that would surfeit the voracity of the most persevering book worm; and it goes on increasing and multiplying in a manner that makes one long for a Malthus to preach some degree of moderation to its producers. All must be read, however, lest some grains of wheat should be thrown away with the chaff.

If this be multiplied by fifteen (the number of sections into which this congress is divided), the enormous literary output alone justifies specialism in part of it. Much original literature has been published in *The Journal of Laryngology*, which was founded in 1887 by Mackenzie in association with Dr. Norris Wolfenden.

While the trend in modern times has been towards ever-increasing specialism, less time has been available for research. Yet the written record of such research is the most worthy monument that lives after us. It becomes apparent that, if research is to be pursued wholeheartedly, private practice and the income therefrom must be relinquished. While the salary of a Hollywood star can never be envisioned in this regard, a salary of little more than the basic wage is an insult to what is generally regarded as the highest type of intelligence, that of a research worker. The resolutions of the National Health and Medical Research Council (THE MEDICAL JOURNAL OF AUSTRALIA, March 6, 1937) include *inter alia* a recommendation that "from time to time Senior Research Fellowships should be available at a salary of not less than £1,000 *per annum*". Yet the Federal Cabinet in March, 1937, declared that "for the present it is not possible to guarantee the money which would be needed to launch the project" (*The Sydney Morning Herald*, March 11, 1937). Eventually the *Medical Research Endowment Act*, 1937, was passed, but unfortunately no sum of money was mentioned in the Act. Should the scheme later come to fruition, however, members of this section could well grace such positions and so shed lustre on a branch of medical science so dear to the heart of Mackenzie.

During the last year of his life Sir Morell devoted himself very actively to his literary work, until in November, 1891, he contracted influenza, of which he died on February 3, 1892. His name will live as an outstanding figure of his day, a remarkable writer who left his impress on the present century, an excellent teacher, a great organizer, a pioneer in rhino-laryngology, and a surgical specialist of international repute. The lines from Momerie's "Immortality" seem to breathe the spirit of the man:

> The facts of life confirm the hope
> That, in a world of larger scope,
> What here is faithfully begun
> Will be completed—not undone.

SOME ASPECTS OF GOVERNMENT CONTROL OF TUBERCULOSIS.[1]

By H. W. WUNDERLY,
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YEARS ago, in almost all countries, the control of tuberculosis and the fight against its spread were left in the hands of a few public-spirited bodies. It soon became universally recognized that the magnitude of the problem was such that private enterprise could not handle it alone, and the assistance of the government became essential.

To be successful there must be close cooperation between governmental and non-governmental agencies. It was necessary to have two kinds of legislation for the effective control of this communicable disease. Firstly, there had to be proper provision for the raising of the necessary funds for the fight against tuberculosis. Secondly, to prevent the spread of infection the reporting of cases of tuberculosis had to be compulsory, and there had to be very definite control of such cases.

It was undoubtedly the pioneering work of the various philanthropic organizations which showed the various governments the right methods in combating this contagious disease in its public aspects.

Nathan Raw, in his presidential address delivered before the Hunterian Society in 1932, recalled that

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

although Koch discovered the tubercle bacillus in 1882, it was not till 1908 that he was convinced that bovine tuberculosis could be conveyed to man.

In 1918, in the United States, an experiment was started in which an attempt was made to wipe out all tuberculosis in cattle by slaughtering every animal with any trace of the disease. At that date the incidence of infection was about 5%. In the year 1931-1932 nearly 13,500,000 cattle were tested, and only 1·9% gave positive reactions. This campaign, at the start, met with a good deal of opposition, for it meant the destruction of a great many cattle apparently in perfect health, and the compensation paid seldom approached the value of the live animal.

The authorities in England recognized the value of the results of this experiment, but they felt that the financial burden would be too great. It has been stated that while about 40% of the cattle in Great Britain are tuberculous, only between 1% and 2% have tuberculosis of the udder and about 8% of the milk (in 1932) contained the tubercle bacillus.

Jensen,[6] when investigating the incidence of bovine infection amongst the cases of human tuberculosis in Denmark, found percentages varying from 6 to 28 amongst cases of pulmonary tuberculosis in children up to fifteen years of age, 2 to 16·9 in people between fifteen and thirty years, and up to 6·7 in people over thirty years.

In a report on bovine pulmonary tuberculosis in man, Tobiesen, Jensen and Lassen[14] investigated 26 cases. Ten of these patients were under five years old, and none were over thirty-two years. In 13 of these cases the patients had been drinking raw milk for some time, while only three denied that they had ever taken raw milk. Five of the ten children under five years of age had previously shown abdominal symptoms that might be interpreted as due to abdominal tuberculosis. The pulmonary processes that were demonstrated by X ray examination appeared not to show any particular features that would differentiate them from the changes characteristic of the respective age classes as generally seen in pulmonary tuberculosis.

They concluded that:

> It seems evident that the prognosis of pulmonary tuberculosis in the age class under five years is serious, probably just as serious as the prognosis of pulmonary tuberculosis due to the human type of the bacillus.

Dr. R. Webster and Dr. W. J. Penfold reported to the Australian and New Zealand Association for the Advancement of Science in Melbourne in 1935 that they had found that 25·9% and 22% non-pulmonary lesions in children yielded the bovine organism, and several workers in England have obtained similar results.

The Commonwealth Government is taking an interest in this matter, and in this State Dr. Platt has commenced to group the types of tubercle bacillus. At present, very few figures are available, but sufficient work has been done to show that the bovine organism is responsible for much invalidity.

There are two methods of attacking this problem of bovine infection in human beings: (*a*) eradicate tuberculosis in cattle, and/or (*b*) pasteurize all milk.

The authorities in the United States of America regard the huge sum of money paid in compensation as a very sound investment, and, as soon as the farmers realized that the healthy members of their herds were being protected by the destruction of the diseased animals, they gave their full support.

In the State of South Australia the only cattle which are compulsorily tested for tuberculosis are the "subsidy" bulls. There is no tuberculin testing of dairy cattle except at the request of the owner, and as all positive reactors have to be slaughtered, without any compensation, the requests for such tests are very few.

It might pay the various Governments of the Commonwealth to insist that all milk be pasteurized, but for it to be effective it must be done properly. The question of pasteurization of milk has recently been discussed in the House of Lords, and recent experiments have shown that "milk suffers no damage by pasteurization that is important compared with the risks of drinking it raw".

While it has been recognized for many years that cattle can be prevented from infecting human beings with the tubercle bacillus by slaughtering all the diseased animals, it took a long time for it to dawn upon the medical world and the public health authorities that human beings with tuberculosis could be prevented from infecting healthy people. This does not necessarily mean complete isolation of the patient, for most people can be taught how to prevent infection from spreading to those around them.

The importance of segregating the patient with advanced and infectious pulmonary tuberculosis has long been recognized. The majority of such patients are only too glad to avail themselves of the medical care and skilled nursing which they can obtain in a hospital-sanatorium. But for a very small minority it is wise to have an act which provides for compulsory segregation. The 1925 Act of the Ministry of Health in Great Britain contains such a clause, but it is seldom necessary to make use of it. The knowledge of the power of compulsory removal is usually sufficient to ensure compliance.

In the anti-tuberculosis campaign it is important to prevent, as far as possible, the passage of tubercle bacilli from patients to people with whom they may come in contact. So it is essential to find the open case and to prevent it from acting as a source of contagion to others.

Having found the "open" case, an attempt should be made to convert it into a "closed case", that is, one which cannot disseminate tubercle bacilli.

For the campaign to be efficient there must be facilities for treating these seriously ill patients. A few years ago the sanatoria were kept for those suffering from early mild infections, who would benefit by ambulatory treatment and who were

afebrile. Especially with the extension of the uses of the various methods of collapse therapy, the use of sanatoria and hospital-sanatoria has been extended. Under the old method each bed would be occupied by one or two patients a year. Better use can be made of these beds by having a quick turnover. An attempt is made to convert each patient from an "open" to a "closed" case. It is the presence of the "open" cases in the community that is keeping tuberculosis a current problem amongst us. By the use of the various methods of lung collapse we attempt to stop the output of tubercle bacilli. These patients, when discharged from the sanatorium, are not cured, but they are now much less of a menace.

It is false economy to discharge a patient who is doing well, but whose condition is not quite quiescent, to make room for a newcomer. While it is not necessary to keep him in the hospital-sanatorium, it is necessary to put him somewhere where his progress can be carefully observed. Such a place is a village settlement. So the ideal arrangement is to have the hospital, sanatorium and village settlement all together.

The ideal sanatorium should deal with the disease in all its stages and have its hospital for its absolute rest cases, both those in advanced and those in acute stages of the disease, and those requiring surgical treatment; its pavilions for those who are fit for graduated exercise; and its village settlement for those who are convalescent and able to work.

In the fifteenth annual report of the Ministry of Health the Minister suggested that an institution of less than 100 beds was unlikely to be able to provide the most efficient treatment in the most economical manner. The Joint Tuberculosis Council suggested a 250 bed unit as the optimum size for a combined institution.

For maximum efficiency in the campaign against tuberculosis there must be a "sufficiency of facilities for treating the most seriously ill of the patients and those unable to provide for themselves or to follow the directions for preventing the spread of infection".[5]

As the result of a survey in Massachusetts, a relationship was discovered between annual deaths and existing active cases, and from this it was possible to set up a standard for the number of beds needed for tuberculosis. This standard was fixed at one bed for each death annually from tuberculosis.

Crocket,[4] in 1934, reviewed the world figures between 1929 and 1932. He found that adequate provision of institutional treatment is almost invariably associated with a correspondingly low death rate from tuberculosis.

A. Those countries with a low death rate—death rates ranging from 46 to 90 per 100,000—have an average of 0·97 bed per death.

B. Medium death rates—death rates from 91 to 133 per 100,000—have an average of 0·40 bed per death.

C. Those countries with high death rate—death rates from 161 to 199 per 100,000—have an average of 0·20 bed per death.

In South Australia we have just over one bed per death—245 beds and 235 deaths in 1936—while in 1932 there were only 153 beds to 275 deaths. We are finding one bed per death very inadequate, and would like 1·5 to 2·0 beds per annual death. Chadwick[3] was so impressed with the results of the standard in America that in 1933 he suggested a new standard of two beds per annual death, and hoped by that means to make tuberculosis a rare disease in those communities making such provision.

Time will not allow me to deal at any length with the question of the provision of "after-care" for patients who have passed successfully through the hospital or sanatorium or through both.

I just want to repeat that it is uneconomical until his disease is quite arrested to return a patient to the conditions of home and employment under which his health originally broke down. Also, as Varrier Jones has so frequently stated, it is foolish to expect convalescent patients to be able to compete in the open labour market.

If the plan of attack were to wait till the patient with positive sputum presented himself for examination, we should have fulfilled our mission if we were to do everything possible to change these "open" to "closed" cases; and if that were not possible, to prevent them from spreading infection. This is a very important part of the campaign, but it is very humiliating to find that the prognosis of the individual case has been but little improved by the use of modern methods of treatment.

D'Arcy Hart,[2] in his recent Milroy lectures, stated that:

The decline in mortality is probably due to the decrease in incidence of new cases rather than to a reduction in the case fatality. In other words, in spite of the advance in treatment, the prognosis has not been materially affected; but mainly owing to the improved social and economic conditions of the mass of the people there has been a reduction in the number of new cases.

Hartley, Wingfield and Burrows, from reports of the Brompton Hospital Sanatorium, Frimley, conclude that the prognosis of an average case of pulmonary tuberculosis has not materially changed during the past thirty years, though that of a small selected class, treated by collapse therapy, has considerably improved. Despite this, Dr. Otto May, speaking for the medical officers of life assurance companies, would not admit that the use of an artificial pneumothorax or of thoracoplasty materially improved the "risk" from the companies' point of view.

It is generally accepted that prognosis is related to the stage at which the disease is recognized. The apparent lack of improvement in average prognosis suggests that the proportion of cases diagnosed in the slighter or earlier stages has not materially increased. This is borne out in the fifteenth annual report of the Ministry of Health, to which previous reference has been made. The Minister states:

The fact that for the past ten years there has been no increase in the proportion of notification of new cases of pulmonary tuberculosis to the number of deaths occurring from pulmonary tuberculosis, suggests that (contrary to the findings in the case of non-pulmonary forms of tuberculosis) there had been no increase in the proportion of patients notified in the earlier stage of the disease. This had been due to delay on the part of the patient in seeking medical advice.

We cannot afford to wait till patients seek medical advice; we must go out and find them. Tuberculosis is of such insidious onset and course that patients in general do not seek advice until late in the disease. This "case finding" can be tackled effectively in two ways: (i) by the examination of all contacts of cases of known disease, (ii) by an investigation of whole communities.

It is in this sphere that the work of the visiting nurse is invaluable. Not only must she render assistance to the patient and act as teacher to the household, but she must investigate the home conditions and persuade all the contacts to come up to the tuberculosis clinic for thorough investigation.

Opie and McPhedran,[1] who took part in the investigation of a thousand families in Philadelphia, state that:

Since the disease occurs in large part as long continued household epidemics, the household or family should be made the unit for observation of a prophylactic case.

In the same report they recommend the annual examinations of all members of households of which some member suffers with tuberculosis. And when children or adult members of such a household have respiratory infections between routine examinations, X ray photographs are taken of their lungs.

Whether the case finding be conducted in the households of people suffering from tuberculosis or amongst families, the family being the unit for investigation, or in schools, universities or industries, the procedure is the same. The first part of the investigation consists in taking a careful history, which should include an inquiry into home conditions, place of employment, contact with illness *et cetera*. Then, after a thorough clinical examination, a Mantoux test should be done.

It would be an advantage if a standard tuberculin were used in this test. Investigations conducted at the Henry Phipps Institute showed a wide variation in preparations of "O.T." from various manufacturing companies and various state and government laboratories. The essential requirements for a standard tuberculin for general use are specificity, constant potency, stability and non-sensitizing nature. The methods of Seibert[7] enable the isolation from "O.T." of a protein derivative with these characters. A series of tests on 2,000 subjects indicates that the respective dosages of 0·00002 and 0·005 milligramme for first and second injections will avoid severe reactions, and at the same time detect approximately all of those cases detected by the best preparations of old tuberculin. This protein derivative is now supplied in tablet form with a supply of diluting fluid, so that 0·1 cubic centimetre delivers the required dose.

To perform the test, 0·00002 milligramme is injected intracutaneously, and the result is read in forty-eight hours; if there is no reaction, 0·005 milligramme is injected into the opposite arm. If this, too, gives no reaction after forty-eight hours, the result of the test is negative. The deleterious effect of age upon diluted tuberculin has not been fully appreciated, despite the fact that as far back as 1909 Mantoux noted that its potency decreased upon standing. Aronson[8] quoted Okell, who observed that a dilution of 1 in 1,000 of "O.T." kept at room temperature loses 40% of its potency within ten weeks; and Aronson found that a dilution of 1 in 10 "O.T." kept at 37° C. loses an appreciable amount of its potency within seven to ten days, and that after several months at this temperature very little of its activity remains.

The results of testing with "P.P.D." are recorded as follows:

"+": redness *plus* definite œdema, 5 to 10 millimetres in diameter.
"++": redness *plus* definite œdema, 10 to 20 millimetres in diameter.
"+++": redness *plus* definite œdema, over 20 millimetres in diameter.
"++++": redness, œdema and an area of necrosis.

If there is no swelling at the site of injection, the result of the test is read as negative.

I dwelt at some length on this Mantoux test because it forms such an important part of the investigation of contacts and of case-finding generally. Opie and McPhedran found that:

A negative tuberculin reaction in persons attending a dispensary clinic indicates the absence of any significant, that is, unhealed tuberculous lesions, with a possible error of one in more than 500 instances.

Also they discovered that:

In young persons of less than twenty years of age exposed to familial contact with tuberculosis, the incidence of lesions increased with the increasing intensity of the tuberculin reaction.

The absence of a reaction to the Mantoux test is presumptive evidence that there is no tuberculous infection. With complete recovery from tuberculosis, sensitization disappears and calcified nodules of healed tuberculosis may be found in lungs and bronchial lymph glands in the absence of the tuberculin reaction. Long, Siebert and Aronson[9] found that:

X ray examinations indicated that purified protein derivative, like other tuberculins, may fail to detect an occasional case of healed primary tuberculosis with calcified foci, but that cases of clinical significance are not missed.

Thus the Mantoux test can be used for separating those being investigated into two definite groups, and those failing to react need not be submitted to an X ray examination. Later I shall refer to those failing to react who are in contact with cases of tuberculosis.

In the investigation of children, parents frequently object to any kind of injection, and so an attempt is being made to see how the Moro test compares in accuracy with that of the Mantoux

test. Sir Robert Philip has used tuberculin ointment for many years and found it satisfactory. Another difficulty with a large scale campaign is the cost of the X ray examinations. We have nothing to compare with the Powers X Ray Tuberculosis Travelling Survey Service, which can take 100 to 150 photographs an hour at a cost of about three shillings a head. These paper films are used for examining men enrolling for the Civilian Conservation Corps camps in the United States of America and in many large surveys in that country. Perhaps it would be possible for the various governments in this country, by buying in large numbers, to obtain the celluloid films at very reduced rates.

If the absence of funds makes it quite impossible to make use of X ray photography, about 90% to 95% of lesions can be detected by the use of fluoroscopy. Reid,[10] of the Metropolitan Life Assurance Company, in describing five years' experience in fluoroscopy says:

> By reason of its accuracy, economy, rapidity and simplicity of technique, it is the most efficient basis of selection for complete roentgenographic examination.

And Dr. V. Romberg, in Germany, proved that 95% of all the cases detected by Röntgen ray pictures can also be diagnosed by fluoroscopic examination. Reid further stated:

> We are detecting tuberculosis in the early states: sanatorium admissions in 1928 were 42% minimal lesions; in 1932, 76% minimal.

It has been discovered that on the average five years elapse between the first X ray evidences of pulmonary infiltration and death.

Another highly important fact is that, also on the average, clinical symptoms do not occur till some three years after early pulmonary infiltration can be recognized by X rays.[11] With the hope of improving the prognosis, the importance of early diagnosis has been stressed. It is no good waiting till symptoms have developed; the diagnosis must be made in the pre-clinical stage. I again stress that we must not wait for the patient to come to us; we must go out and find him.

A few years ago most anti-tuberculosis campaigns tackled the problem of childhood infection. While we still recognize the importance of this sphere of activity, we now realize that the important ages are from fifteen to twenty-five years. Almost all over the world there has been a rapid decline in the mortality from tuberculosis; but in this age period, especially for the female sex, there has been an inexplicable halt in the march of progress. So it is to the adolescent and to the young adult that we must direct our attention. If the governments hope to control tuberculosis, they must make arrangements for the systematic examination of these valuable members of the community. Long writes:

> Many physicians are apparently still unaware that pulmonary tuberculosis is essentially symptomless in its origins, or at least begins with symptoms no more characteristic of it than of other respiratory infections, and is very commonly devoid of physical signs.

During the period when the disease is minimal, but slowly progressing, it may be detected by X rays if not by physical signs. So we should turn our attention, in the first instance, to the youths and maidens of high school age, to the undergraduates of our universities, to the shop assistants in our big emporiums. Later on, it might be possible to extend the survey to the big industries, to the government services, and to the recruits for the army, the navy and the air force. Under present conditions it is impossible to make a complete survey of the whole community, although this is part of the plan adopted by the Federal Health Council for a Commonwealth-wide campaign to combat tuberculosis. Either a survey must be made of families, as was done in Philadelphia by the staff of the Henry Phipps Institute, or a complete survey made of school children in the period fifteen to twenty years, and a start should be made to examine all university undergraduates and scholars of technical colleges.

In conclusion, I want to try to solve the riddle of the "tuberculin negative" individual. If the individual be not in contact with known cases of tuberculosis, it should be sufficient to repeat the Mantoux test at twelve-month intervals. If, at a subsequent examination, the response is found to have changed from negative to positive, the present opinion is that the subsequent examination should be at more frequent intervals. The real difficulties arise when we have to consider the position of the person failing to react who is compelled to remain in contact with a case or cases of tuberculosis. Heimbeck's experience at Oslo has been confirmed by several observers. Paretzky,[12] in an article on the intracutaneous reaction, stated that a comparison of numerically almost equal groups of positive and "negative" tuberculin reactors that belonged to the same families, showed that both diagnosis and prognosis were considerably more unfavourable in the non-reacting group. The non-reactors that remained non-tuberculous were either removed from the source of the infection or had acquired a positive tuberculin reaction. Special attention is paid to non-reactors if, for some reason, they remain exposed to infection; they are frequently reexamined and otherwise closely watched. Mariette[13]

> has also provided tentative confirmation of the greater risk of the adult type of disease appearing in this group [of negative reactors] than in the group of originally positive reactors.

This has not been the experience of the physicians in charge of the student health services at Pennsylvania, and it is in direct conflict with the findings of Opie in his home-contact series.

It remains to be decided what should be done with nurses, medical students and hospital attendants who, being "tuberculin negative", are compelled to be in contact with cases of tuberculosis. Heimbeck, at Oslo, and Gravesen, in Denmark, have had very good results from vaccination with "B.C.G." vaccine. Those thus vaccinated were not

allowed to come into contact with tuberculosis till they had become "tuberculin positive". Individuals with a positive reaction who later develop the adult type of tuberculosis may be the victims of an exogenous or of an endogenous super-infection. Opie and his followers favour the former possibility, while Pagel is definitely of the opinion that many are endogenous in origin. All are agreed that the ideal is to prevent infection in every form, be it milk-borne or sputum-borne.

So government control must aim at stopping the spread of infection, be it milk-borne or sputum-borne. It must hunt down the open case, and either convert it into a closed case or prevent, by educative means, its spreading infection. Further, it must seek out the early cases in the pre-clinical stage and take such measures as have been found to be effective to protect those who, with a negative reaction to tuberculin, are compelled to be in contact with tuberculosis.

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SILICOSIS AMONG METAL MINERS IN WESTERN AUSTRALIA.[1]

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OPPORTUNITIES for studying the clinical types of the various pneumonokonioses are found in all mining centres, but those for observing accurately the progress of industrial disease are not found in many places.

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

At Kalgoorlie, Western Australia, however, such an opportunity does exist on account of the fact that the legislation governing the mining industry has required for some years, firstly, the certification after clinical and radiographic examination of all mine workers of freedom from pulmonary disease prior to employment in the industry, and the reexamination, also by radiograph, at regular intervals of all workers in the industry.

This legislation came into force in 1925, and, by arrangement between the Federal and State Governments, the medical examinations rendered necessary thereby have been carried out since that time by the staff of the Commonwealth Health Laboratory, Kalgoorlie. Consequently the records of this institution contain a great deal of valuable material for inquiry.

The first procedure in this connexion was the making of a survey, all mine workers in the areas affected by the proclamation of the Acts being examined. After the employees of the mines in and around Kalgoorlie had been examined, a tour of the outlying mining centres was made by a party equipped with a portable X ray plant and laboratory. This survey included a thorough clinical examination and a chest radiograph of all men examined. The report has been published by the Commonwealth Department of Health [Service Publication (Division of Industrial Hygiene), Number 5]. Since the end of 1926, which marked the completion of this piece of work, employees in the Kalgoorlie district have been reexamined annually, and visits at intervals as short as practicable have been paid to the outlying centres for the same purpose.

In making this inquiry it has been realized in the first place that radiography has taken immense strides in the period under review and, secondly, that individual observers do not invariably see eye to eye in matters of diagnosis. Accordingly the clinical records and all the serial radiographs of the subjects of the inquiry have been examined by the writer personally. In the case of the radiographs due allowance has been made for improvement in technique, and the original diagnoses were modified when considered necessary in the light of present-day knowledge and experience.

Effect of Legislation on the Health of the Industry.

The provisions of the *Miners' Phthisis Act*, 1922, of Western Australia, now embodied in the *Mine Workers' Relief Act*, 1932, require the elimination from the industry and compensation of all workers who are found to be suffering from tuberculosis, whether complicated by silicosis or not. The latter Act also provides for total compensation of those men found to be suffering from advanced silicosis, contingent on their leaving the industry. The regulations of the *Mines Regulation Act*, 1906, which were promulgated in 1926, require certification of freedom from tuberculosis as well as any degree of industrial pulmonary fibrosis and certain other diseases in the case of all men prior to entering the industry. By these means the mines are kept

free from tuberculous infection and a healthy body of recruits to the industry is assured. Table I speaks for itself in showing the cleansing effect of these legislative provisions. It should be noted that the columns headed "Normal and Anteprimary Silicosis" and "Silicosis Only" represent an actual statement of the percentages of these conditions in the industry year by year, while the two following columns showing tuberculous cases represent an annual incidence owing to the elimination of cases as they occur.

TABLE I.

Percentage of Normal and Silicotic Cases from Year to Year and Annual Incidence of Tuberculosis, 1925 to 1936.

Year.	Normal and Anteprimary Silicosis Percentage.	Silicosis Only Percentage.	Silicosis with Tuberculosis Percentage.	Tuberculosis Only Percentage.
1925–1926 (initial survey)	80·1	16·1	3·5	0·3
1927	80·6	15·6	3·5	0·3
1928	85·5	13·3	1·1	0·1
1929	82·2	15·9	1·6	0·3
1930	82·0	13·3	3·3	1·4
1931	84·0	13·3	1·9	0·8
1932	89·2	10·2	0·4	0·2
1933	86·5	13·0	0·4	0·1
1934	92·4	7·3	0·2	0·1
1935	92·3	7·5	0·1	0·1
1936	93·7	6·0	0·2	0·1

The above figures have been taken from the recorded diagnoses year by year without modification. Actually at the time of the original survey, on account of the limitations of that time in chest radiography, the percentage of "Normals, etc." in the industry was a good deal lower and that of silicotics higher.

Diagnosis of Industrial Pulmonary Disease.

The diagnosis of pulmonary fibrosis in its earlier stages depends practically entirely on the chest radiograph.

For routine work of this description in an institution in which many thousands of men are examined annually, considerations of time and expense preclude the full examination to which a private patient would be subjected by the consulting radiologist, and a single film only is taken. It will be appreciated accordingly that it is essential not only to be able to produce radiographs of the highest standard for this purpose, but also that the standard should be uniform for purposes of comparative examination. Until 1929 at Kalgoorlie chest radiographs were taken on small films (ten inches by 12 inches) at a target-film distance of three feet, using 60 milliampères of current, and required an exposure interval of one and a half to one and three-quarter seconds. At the present time, using faster films and intensifying screens and a modern plant, exposures of only two- or three-twentieths of a second are necessary at a target-film distance of six feet and using 250 milliampères of current. This technique represents only about one-third of the total capacity of the plant and is adopted for reasons of economy. Films measuring 14 inches by 17 inches are used, and a much more careful routine of developing produces radiographs of a quality vastly superior to those produced ten years ago. The effect of cutting down the time of exposure is most pronounced in reducing the blurring of lung shadows from the movements of involuntary respiration, shivering and other bodily movements, and from the cardiac pulsations. By this means the fine details of the lung shadows are more easily studied and more accurately evaluated. Thus, while the radiograph of 1925 reveals with certainty a gross tuberculous lesion or a coarsely mottled silicotic process, the fine ramifications of the bronchial tree and mild grades of pulmonary fibrosis were distinguishable only in a small proportion of films.

The report of the International Conference on Silicosis held at Johannesburg in 1930 [International Labour Office Studies and Reports, Series F (Industrial Hygiene), Number 13, page 88] gives the following criteria in connexion with the diagnosis of silicosis:

For the diagnosis of silicosis as a disease it is necessary to take into consideration: (*a*) the employment history, (*b*) the symptoms and physical signs, (*c*) the radiological findings.

The disease can conveniently be divided into three stages, designated "first", "second" and "third" stages.

In the differential diagnosis of silicosis from other pneumoconioses a history must be established of exposure to silica dust in a quantity reasonably commensurate with the clinical and radiological findings.

In the "first stage" symptoms referable to the respiratory system may be either slight or even absent. Capacity for work may be impaired. There may be a departure from the normal in percussion and auscultatory signs, and the radiograph must show an increased density of linear shadows and the presence of discrete shadows suggestive of nodulation.

In the "second stage" there is an increase of the physical signs observable in the "first stage", and the radiograph shows an increase in the number and size of the discrete shadows indicative of nodulation with a tendency to their confluence. There must be some degree of definite impairment of working capacity.

In the "third stage" all the above conditions are grossly accentuated and indications of areas of massive fibrosis are usual. There is serious or total incapacitation.

Pulmonary tuberculosis may be present in any of the above described "stages of silicosis", altering the symptoms, physical signs and radiographic appearances and the degree of working capacity.

Radiographs may frequently be met with which show a slight, moderate or well marked increase beyond the normal in radiating linear shadows. These may or may not be due to fibrosis.

The above statement, formulated seven years ago, still serves as the basis on which the diagnosis of silicosis and the estimation of its degree of advancement is made. At that time the radiographic appearances of other pneumonokonioses, such as anthracosis, were hardly known. Study of present-day radiographs of mine workers permits an elaboration of the foregoing data, although admittedly the difficulties in the way of a simple classification are increased.

In Western Australia gold mining is conducted over many thousands of square miles of country in many different types of country rock and under varying underground conditions. In addition to this miners are wanderers as a class, moving from field to field, and it is the exception far rather than the rule to find that one man has spent his industrial life in one mine or, for the matter of that, in one State or country. Consequently it is difficult to mention a particular type of fibrosis, radiographically speaking, as characteristic of one mining area, although there are certain indications that some underground condition, probably the mineral composition and concentration of the dust inhaled, does influence the type and chronicity of the fibrosis produced.

The Radiograph of the Normal Chest.

In selecting recruits for the industry by radiographic examination it is first necessary to know what constitutes the radiographic appearance of the normal chest. In actual fact the normal chest radiograph is rarely if ever seen, but some working standard must be adopted. The radiograph, as previously stated, should be taken with as short an exposure interval as practicable and should be of the "soft" rather than the "hard" type so that the vertebral shadows do not show individually through the cardiac shadow. Under-penetration, however, producing radiographs of too "soft" a nature results in undue accentuation of the peribronchial shadows in normal individuals.

In the modern radiograph of a normal young adult the pulmonary fields are free of any asymmetrical or pronounced shadows and exhibit the ramifications of the bronchial tree as fine linear markings whose outlines diminish in clarity as they approach the peripheral limits of the fields.

The hilar shadows may vary in size and density within the limits of normality and may show relatively dense areas of glandular or other elements which are not considered as being clinically significant.

On the mesial aspects of both fields the linear markings of the larger bronchial divisions are broader and more clearly apparent both above and below the hilus, the basal shadow on the left side, however, being largely or totally eclipsed by the cardiac shadow. At the extreme apices, also, above the clavicles markings are frequently observed suggestive of scarring of very small, old lesions or of a slight degree of pleural thickening. These are common in the robust as well as in persons of lesser physique and are not associated with any clinical signs capable of detection and are accordingly not regarded as significant.

Other isolated areas of slightly increased density in the radiograph which cannot be confirmed by clinical observation are seen in a proportion of cases. These are most commonly situated in the subapices, although sometimes observed near the hilar regions or at the bases, and their significance is not understood. Probably they are small fibrotic areas which are the residue of old infections, whether tuberculous or pneumonic. Their possible influence on the development of silicosis will be discussed later.

In a series of 1,180 "normal" men examined at Kalgoorlie, markings of this nature were observed in 83 or 7·0% among old films, while among 420 normal persons examined in 1936, that is, when modern apparatus and technique were used, 191 or 32·4% showed these shadows.

In young adults and adolescents a generalized accentuation of the peribronchial markings of a soft and fluffy appearance with indistinct outlines is frequently observed radiographically and is most common in mouth breathers and sufferers from nasal catarrh or sinusitis. These markings are regarded as being catarrhal in origin and the experienced observer is not likely to confuse them with the appearance of an early pneumonokoniosis. Out of a series of 500 consecutive men examined at Kalgoorlie, 105 or 21·0% showed radiographic markings considered to be catarrhal in type. The average age of these men was 25·1 years as against 30·0 years for the rest of the group.

As age advances, even in the absence of exposure to industrial dust, the peribronchial markings show evidence of fibrosis by means of a uniform accentuation of a clear-cut and well delineated type. Due allowance must therefore be made for age in the estimation of the normal chest radiograph.

In general, also, workers in out-door occupations not necessarily exposed to excessive dust inhalation show more definite bronchiolar markings than, for instance, those who have worked always in an office.

Early Pulmonary Fibrosis.

The early effects of exposure to the inhalation of dust while at work are evidenced radiographically by an increase in the density and size of the peribronchial shadows. This can be demonstrated in the radiographs of workers in many industries. Stokers, metal workers, farm workers and others are frequently shown to have a degree of pulmonary fibrosis in excess of normal.

In mine workers exposure to siliceous dust appears to cause a fibrosis of two distinct radiographic types. In the first type, which is more commonly observed in machine miners engaged for some years in stoping or development work, the peribronchial markings are accentuated in an exceedingly symmetrical and well-ordered manner. In the second type, which is found commonly in surface workers, the radiographic markings are less regular and present a more confused appearance, as well as being broader and less defined in outline. What factors influence the development of these two types is not at present known, although there are indications that latent infection may play its part.

Table II shows the distribution of these radiographic types of fibrosis in accordance with class of occupation among 833 cases of early pulmonary fibrosis studied. This grade of fibrosis corresponds with the diagnosis of "ante-primary silicosis".

TABLE II.

Distribution of Radiographic Types of Early Pulmonary Fibrosis in Mine Workers according to Class of Occupation.

Class of Occupation.	Total Cases.	Type 1.		Type 2.	
		Number.	Percentage.	Number.	Percentage.
Underground ..	343	246	71·7	97	28·3
Surface ..	355	209	58·8	146	41·2
Mixed	135	93	68·9	42	31·1
Total ..	833	548	65·8	285	34·2

Clinically it is exceedingly difficult to detect signs of this degree of fibrosis and there is rarely, if ever, any degree of incapacitation attributable directly to the condition.

Early Silicosis.

As industrial pulmonary disease advances, the linear markings observed radiographically in the ante-primary stage commence to break up and the well-known mottled appearance of the lung fields commences to show, corresponding with the formation in the lungs of the miner of the characteristic nodules of silicosis. Concurrently, on the clinical side there is usually at this stage some degree of impairment of health associated with dyspnœa, cough and diminished capacity for work and, to the stethoscope, signs of diminished air entry into the lungs.

Here again, in the observation of the cases under review, one readily distinguishes two types of fibrosis which appear to correspond with the two ante-primary types. In the first type the mottling is fine, and in the second, coarse.

Table III shows the relative frequency of these types in a series of 547 cases of early silicosis.

TABLE III.
Distribution of Fine and Coarse Mottling in Radiographs of Mine Workers with Early Silicosis according to Occupation.

Class of Occupation.	Cases of Early Silicosis.	Fine Mottling.		Coarse Mottling.	
		Number.	Percentage.	Number.	Percentage.
Underground ..	414	325	78·5	89	21·5
Surface ..	34	23	67·7	11	32·3
Mixed	99	88	88·9	11	11·1
Total ..	547	436	79·7	111	20·3

Advanced Silicosis.

The stage in the progress of industrial pulmonary disease known as advanced silicosis is marked by clinical rather than radiographic changes. The subject complains of severe dyspnœa and is unable to continue at strenuous work. There is always cough with sputum, frequently blood-stained, and evidence of commencing failure of the right heart.

Radiographically there may be little or no difference from the picture of early silicosis, but frequently it is noted that the mottling becomes denser and the individual mottles tend to coalesce, giving the radiograph a blotchy appearance.

Table IV gives the distribution of radiographic types of mottling in 161 cases of advanced silicosis.

TABLE IV.
Distribution of Fine and Coarse Mottling in Radiographs of Mine Workers with Advanced Silicosis according to Occupation.

Class of Occupation.	Cases of Advanced Silicosis.	Fine Mottling.		Coarse Mottling.	
		Number.	Percentage.	Number.	Percentage.
Underground ..	125	67	53·6	58	46·4
Surface ..	11	4	36·4	7	63·6
Mixed	25	6	24·0	19	76·0
Total ..	161	77	47·8	84	52·2

Silicosis with Tuberculosis.

It is well recognized that active pulmonary tuberculosis may complicate the silicotic process in any of its stages. In actual practice in Kalgoorlie, while the ante-primary or "first" stage is not recognized as actual silicosis for the purposes of notification to the authorities, an ante-primary case which becomes complicated by tuberculosis is diagnosed as "silicosis with tuberculosis".

In this condition the predominant symptoms are those of tuberculosis: cough; sputum; hæmorrhage from the lungs, usually slight; pain in the chest; dyspnœa on slight exertion; loss of energy and weight; and night sweats. The majority of these symptoms are present in uncomplicated advanced silicosis, and the clinical picture of this condition may represent the syndrome of a very chronic type of pulmonary tuberculosis. Hence in many cases it is frequently difficult to decide when tuberculosis is present. The presence of the tubercle bacillus is the exception rather than the rule, except when undoubted tuberculosis supervenes early in the course of industrial fibrosis.

The radiographs of silicosis with tuberculosis are characterized by evidence of the fibrotic process with the usually asymmetrical shadows of tuberculous consolidation. Commonly the upper portions of one or both lungs are infected, but fairly frequently the infection becomes activated in one or both basal areas. Less frequently still an infective process radiating from the hilar region of the lung is observed.

Table V shows the distribution of fine and coarse radiographic mottling among 164 cases of silicosis with tuberculosis according to class of occupation.

TABLE V.
Distribution of Fine and Coarse Mottling in Radiographs of Mine Workers with Silicosis and Tuberculosis according to Occupation.

Class of Occupation.	Number of Cases.	Fine Mottling.		Coarse Mottling.	
		Number.	Percentage.	Number.	Percentage.
Underground ..	124	80	64·5	44	25·5
Surface ..	19	11	57·9	8	42·1
Mixed	21	18	85·7	3	14·3
Total ..	164	109	66·5	55	33·5

Pulmonary Tuberculosis.

Pulmonary tuberculosis affects mine workers in the absence of any fibrosis, exactly as it occurs in other members of the community, and needs no description here. In this survey 10 original cases were confirmed among 2,931 mine workers—a percentage of 0·34.

In compensation legislation pulmonary tuberculosis in mining and other dusty occupations is regarded as an industrial disease.

Progress of Industrial Disease.

It was realized during the course of the present inquiry that the estimation of the progress of industrial pulmonary disease by examination of the records of those already in the industry was open to objection on account of the fact that many of these men, although not showing clinical or radio-

graphic signs, were well on the way towards that stage. Accordingly a group of 447 men was selected, comprising those originally examined in 1925-1926 who had been less than two years in the industry, together with all normal persons admitted to the industry during 1927.

The annual radiographs and clinical records of those remaining in the industry at least until 1931 were examined up to 1936; thus in many cases an estimation of the effects of ten years' or more work in the industry was allowed. Certain men, although examined since 1931, had not spent all their time at mine work.

The composition of this group according to class of occupation is as follows:

Underground miners	267
Surface workers	155
Mixed surface and underground	25
Total	447

In the estimation of degree of fibrotic change in the lungs as radiographically recorded, it must be borne in mind in the first place that the development of silicosis is a very gradual process, which commences, theoretically, with the first exposure of the subject to the inhalation of siliceous dust. The diagnostic standards for the various stages of the disease must be therefore to a large extent arbitrary, especially in so far as the early stages are concerned. Experience in this matter can be gained only by the examination of many thousands of radiographs. Accordingly, in the first place radiographic changes have been graduated in degree only, and the following classification has been adopted: (*a*) nil or very slight, (*b*) slight to moderate, (*c*) considerable, (*d*) infective. The last graduation indicates the development of definite or probable tuberculosis.

Underground work has been divided into "stoping and development work" and "other underground work", the workers including truckers, samplers, timberers, mine officials and others working full underground shifts. Surface work has been classified as "dry" or "other" accordingly as the type of work involves undue exposure to the inhalation of fine dust or not. Mixed workers are those who have spent part of their time underground and part doing surface work.

Table VI shows the radiographic changes recorded in the above group irrespective of actual length of time worked.

TABLE VI.
Progress of Industrial Pulmonary Disease.

Class of Work.	Degree.				Total.
	Nil or Very Slight.	Slight to Moderate.	Considerable.	Infective.	
Underground—					
Stoping and developing	55	65	7	1	128
Other	85	50	2	2	139
Surface—					
Dry	6	12	—	—	18
Other	118	16	—	3	137
Mixed work	19	6	—	—	25
Total	283	149	9	6	447

Diagnostic changes, that is, development of ante-primary or early silicosis or tuberculosis, occurred in this group as shown in Table VII.

According to length of time worked, the incidence of the development of ante-primary or early silicosis among these men was as follows: under four years, 7·5%; from five to six years, 22·1%; from seven to eight years, 28·5%; nine years or over, 37·5%.

The onset of tuberculous disease bore no relation to time factor.

Thus at the end of ten years' work over one-third of mining employees in this group, representing at the outset new and healthy industrial material, were found to be suffering from some degree of industrial pulmonary disease. In underground workers the incidence is higher than the general incidence in surface workers, although that among those in dusty jobs is very high. Under present conditions the class of occupation has not seemed to influence the incidence of tuberculosis.

Among a group of 569 mine workers classed as normal according to review of the radiographs of 1925-1926, the percentage incidence of ante-primary or early silicosis was much higher than in the preceding group, amounting to 37·3 for the whole period and approaching 50 for those who had nine or more years' work after 1925-1926. The incidence among underground workers and dry surface workers was again the highest. The incidence of

TABLE VII.

Class of Work.	Ante-primary Silicosis.		Early Silicosis.		Tuberculosis.	
	Number of Cases.	Percentage.	Number of Cases.	Percentage.	Number of Cases.	Percentage.
Underground—						
Stoping and developing	55	43·0	3	2·4	1	0·8
Other	36	25·9	1	0·7	2	1·4
Surface—						
Dry	10	55·5	—	—	—	—
Other	12	8·8	—	—	3	2·2
Mixed	5	20·0	—	—	—	—
Total	118	26·4	4	0·9	6	1·3

tuberculous infection, however, was not much greater, being 1·6% as against 1·3% in the first group.

The records of a group of 585 workers, diagnosed as suffering in 1925-1926 from ante-primary silicosis, were examined to determine the production rate among these men of definite silicosis, whether early or advanced, and the incidence of tuberculosis.

In the period of ten years under review, 160, or 27·3%, of these workers had developed silicosis and a further 25, or 4·2%, had become tuberculous as well. The incidence was very low among surface workers, being only 7·4% among both these classes, while it ranged as high as 47·7% among underground workers.

The influence of time worked in the development of silicosis from the ante-primary stage for all workers is shown as follows: four years or less, 22·5%; from five to six years, 15·7%; from seven to eight years, 29·6%; nine years and over, 31·1%.

Similarly, the rate of development of silico-tuberculosis has been studied among a group of 276 workers who were suffering from early silicosis in 1925-1926. Of these, 78, or 28·2%, were found to be suffering from tuberculous complication prior to or during 1936. The rates among underground workers doing stoping and development or other work were 30·3 and 28·6% respectively; those engaged on dry and "other" surface jobs 20·0 and 30·0% respectively; while that for "mixed" workers was 22·4%.

In this case length of time worked appears to diminish the likelihood of tuberculous infection, which one might easily expect. The percentage incidence according to time worked reads as follows: four years and under, 37·2%; from five to six years, 48·2%; from seven to eight years, 20·3%; nine years and over, 10·5%.

The percentages shown above represent the infected proportions of each section of the main group who have worked after 1926 for the periods stated.

Discussion.

There can be no doubt that, prior to some time in the neighbourhood of 1930, the limitations of chest radiography precluded discrimination between types of early pulmonary fibrosis, and it is also certain that the finely mottled radiographic type of silicosis was in those days classed as ante-primary in degree. Inspection of such radiographs taken with a modern plant, however, clearly demonstrates the difference between accentuated linear markings and fine mottling. It was first thought possible that this fine mottling was characteristic of very early silicosis and became coarse as the disease advanced. The present inquiry has shown that this is not the case, but that it is possible with experience to predict from the radiographic appearance of the anteprimary stage the type of the ultimate silicotic development in many, if not in the majority, of cases.

Instances are observed fairly often in which a fine silicosis becomes complicated by a coarse mottling which usually becomes apparent at first in one lung or one lobe of a lung starting to spread from one of the anomalous shadows previously described and spreading with comparative rapidity, that is, in one or two years throughout both lungs until a symmetrical radiographic picture results. The fine mottling may still be seen underlying this process.

In the majority of cases, however, a silicosis originally of the fine type remains fine even in the presence of tuberculous infection, while the mottles of the coarse type may usually be seen developing like faint and widely distributed ghosts from the second untidy type of the ante-primary stage.

In 1930 the writer in company with Dr. Charles Badham examined radiographically a series of coal miners from the south coast of New South Wales. A large percentage of these men were found to have a well-developed fibrosis closely resembling the fine type of silicosis seen in Kalgoorlie gold miners. When examined clinically these men were surprisingly free from symptoms or incapacitation. They were all men of long experience working in a coal seam in which there is no appreciable silicon content. This condition of pure anthracosis appears to be non-toxic. (The report of this investigation appeared in *Health*, Volume IX, Number 5, page 33.)

In the Western Australian experience of the writer, metal miners who develop the fine type of silicosis also appear to be relatively free from toxic symptoms unless a definite tuberculous complication supervenes, although their incapacity is relatively greater than that of the coal miners.

Metal miners developing the coarse type of silicosis, however, show marked signs of incapacitation fairly early in comparison. This type appears to develop more rapidly than the fine type. Among a series of 325 underground workers suffering from early silicosis of the fine type, 17 or 6·2% had less than ten years' underground experience and 53 or 19·3% less than fifteen years' experience.

Among 89 miners suffering from the coarse type of silicosis the respective percentages were 11·2 and 26·9. Furthermore, many of the conditions designated as coarse silicosis with longer service were originally fine silicosis with an acute coarse formation supervening.

Inspection of a series of radiographs of Bendigo gold miners showed a very much greater frequency of coarse silicosis than is the case in Kalgoorlie. The writer's personal experience in Bendigo leads him to believe that the rate of development of silicosis in Bendigo is considerably greater than in Kalgoorlie, although comparative figures cannot be given. Two factors may influence this, namely, the high silica content of the Bendigo country rock and the high incidence of tuberculosis in that locality.

The writer is led to believe, therefore, in view of the foregoing points that silicosis with fine mottling is a pure form of silicosis of a very chronic nature which *per se* produces incapacitation mainly by its mechanical effect on the right heart after many

years. On the other hand, the coarse type represents the development of a silicosis modified from the outset by some other factor, presumably infection, and in all probability infection by the tubercle bacillus. It develops more rapidly than the fine type in most cases and is to a greater extent incapacitating, the incapacity being due to toxicity rather than to right heart failure. It is to be realized, of course, that the fine type of silicosis may in a large percentage of cases become the seat of a tuberculous infection and at any stage of its development, but this complication is regarded as secondary to the silicotic process in this case, whereas in the coarse type infection seems to preexist or to develop *pari passu* in most cases.

To prove or disprove this contention by statistical methods is difficult, but it is considered the figures quoted earlier help in this direction.

Reference has been made previously to the presence in a certain percentage of radiographs of anomalous shadows thought to be of infective origin. Table VIII shows the distribution of such shadows observed in the radiographs of normal mine workers and those showing varying grades of pulmonary fibrosis at the time of their initial examination. These figures are taken from old radiographs which do not reveal the same percentage of such shadows as do those of today.

TABLE VIII.

Distribution of Anomalous Shadows according to Pulmonary Condition. (Old Radiographs.)

Pulmonary Condition.	Number of Cases.	Percentage of Anomalous Shadows.
Normal	1,097	7·0
Ante-primary silicosis	634	25·2
Silicosis (fine type)	354	53·1
Silicosis (coarse type)	135	70·4

It will be noted that coarse silicosis gives a considerably higher percentage of these anomalous shadows which are believed to be of infective origin than fine silicosis.

One further point remains to be considered in connexion with the present discussion. In South Africa a definite standard of physique is required by regulation in all who are admitted to work in the mining industry. It has been the experience of the authorities in that country that the phthinoid type is a worse risk from the point of view of future compensation than the robust. The percentage of phthinoid chests according to various grades of pulmonary disease has been ascertained in a series of 1,228 Western Australian miners and is shown in Table IX. In this series the percentages of phthinoid chests among coarse and fine types of silicotics were found to be approximately equal. It will be noted that there is very little difference in the percentages among normals and silicotics without tuberculosis, but that the percentage increases with the introduction of the definite tuberculous element.

TABLE IX.

Pulmonary Condition.	Number of Workers.	Percentage of Phthinoid Chests.
Normal	420	20·5
Silicosis (early or advanced) ..	465	19·6
Silicosis with tuberculosis (proved or probable)	335	32·2
Tuberculosis only	8	50·0
Total	1,228	23·5

With regard to the rate of development of pulmonary disease among mine workers in Western Australia, the figures given earlier speak for themselves. It has been shown that an underground worker fresh to the industry under present conditions has an almost even chance of developing at least an ante-primary silicosis after ten years' work and that a considerable percentage of such workers will be partially or totally incapacitated within twenty or twenty-five years. The lesson to be learned from this is that further work on preventive lines is necessary to make the industry safe from the standpoint of health.

Legislative measures have been successful in combating infection by selection of personnel and also by encouraging the potentially tuberculous in the form of the silicotics to leave the mines, helped by compensation. The figures in Table I show that very clearly. The remaining point of attack is the elimination of fine mineral dust from the air of working places. This problem is the centre of constant attention from all interested persons, and there is no doubt that the dust content of the air in mines at most centres has been materially reduced during the present century by the use of water, ventilation and other means. The fact that it has not yet been reduced to a safe minimum, however, is demonstrated by the continued development of pulmonary fibrosis among mine workers, incapacitating them before they should be forced to retire from work.

Summary.

1. A survey has been made of the clinical records and radiographs of a series of mine workers examined at the Commonwealth Health Laboratory, Kalgoorlie, originally in 1925, 1926 and 1927, and at periodical intervals since that date.

2. Two radiographic types of pulmonary fibrosis have been observed among these workers, one of which is considered to be relatively pure silicosis and the other silicosis modified by a toxic or infective element.

3. Pulmonary tuberculosis is found as a complication of each type, but in the first it is regarded as a secondary factor, while it probably preexists and determines the second type.

4. The progress of industrial pulmonary disease among the various classes of mine workers over a period of ten years has been estimated.

POLYCYTHÆMIA, NEUTROPHILIA AND MYELOGENOUS LEUCHÆMIA.

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Historical.

A CLINICAL syndrome, first described by Vaquez in 1892, characterized by persistent cyanosis, splenomegaly and polycythæmia, soon led to the observation of similar cases in France, England and America. Several papers by Osler in 1903 and 1904 definitely established this blood dyscrasia as a clinical entity. In a review of the literature in 1912 Lucas[1] found 179 records of this affection; many cases of Osler-Vaquez disease have since been recognized. *Polycythæmia vera* was defined by Parkes Weber and Bode[2] in 1929 as follows:

> A disease or morbid condition characterized by well-marked, persistent, relative and absolute polycythæmia due to an excessive erythroblastic activity of the bone-marrow, which in the present state of knowledge appears to be the primary morbid factor in the condition; it is characterized likewise by a persistent increase in so-called blood viscosity and often by a cyanotic appearance of the patient and usually, if not always, by easily felt enlargement of the spleen.

The Blood Picture in Polycythæmia Vera.

The normal adult erythrocyte count varies from 4,200,000 to 6,400,000 per cubic millimetre; 5,500,000 is the average for the male and 4,800,000 is the average for the female (Whitby and Britton). In *polycythæmia vera* the erythrocytes may vary from seven to twelve millions per cubic millimetre, and in one case fourteen million cells were recorded. The hæmoglobin level is increased frequently to between 120% and 160%, though it is not parallel to the erythrocyte count; consequently the colour index is below unity, usually between 0·7% and 0·9%. The erythrocytes may be of normal size and regular shape, though anisocytosis, microcytosis and polychromasia may occur and the presence of normoblasts is often noted. Reticulocytes as a rule are only slightly if at all increased in number.

Apart from the erythroblastic activity in the bone-marrow, there is almost always an increased function of the leucoblastic tissue; this is shown by the leucocyte count, which almost invariably exceeds 10,000 per cubic millimetre, and by a granulocytosis, which may form 75% or more of the leucocytes. Myelocytes are often present; their numbers are increased, and they will be accompanied by myeloblasts in the presence of pronounced bone-marrow activity.

The number of blood platelets is usually increased. The total blood volume may be two or three times greater than the normal. Usually no alteration is found in the bleeding and coagulation times.

Pathology.

Overactivity of the blood-forming tissues is shown by the deep red appearance of the marrow in the long bones. There is an increase in the erythroblastic and leucoblastic tissues and in the megakaryocytes. The spleen is enlarged, sometimes even reaching into the left iliac fossa, mainly on account of engorgement with blood and partly on account of a hyperplasia of the splenic pulp. According to Harrop[3] the liver is palpable in at least 50% of the patients.

The pathological changes in the bone-marrow help best to explain the events which may occur in this disease. Apparently the excitant of the red marrow may also cause an increased activity of the leucopoietic marrow and account for the leucocytosis. If this agent acts for a very long time aplasia may occur; Whitby and Britton[4] observed the development of aplastic anæmia in a patient with *polycythæmia vera* of eight years' standing. An increased leucocyte count is a more frequent association of polycythæmia; in the terminal stage of one case of erythræmia Brieger and Forschbach[5] noted a leucocyte count of 255,200 cells per cubic millimetre which were chiefly granulocytes. Blumenthal[6] in 1901 first described the transition of polycythæmia into myeloid leuchæmia; usually the polycythæmia becomes less pronounced as the leuchæmic phase becomes more so. In long-standing cases there is a greater relative increase of granulocytes than of erythrocytes, and consequently these patients may be considered as having erythræmia associated with a non-malignant type of leuchæmia in which few granulocytes are unripe. Weber[7] suggested the term erythro-leuchæmia for this condition. In the late stages a diminished production of red cells may occur on account of aplasia or of a "crowding out of the erythroblastic tissue" associated with a great increase in the leucocytes and resulting in a classical picture of myelogenous leuchæmia. Naegeli[8] believes that there is an initial stage of polycythæmia in all cases of myeloid leuchæmia, but he rejects the theory that this disease occurs in combination with erythræmia; he regards all these patients as having myelosis with an initial high erythrocyte count. Rarely, however, does the latter disease terminate in polycythæmia; Ghiron and Winter[9] reported the development of a pronounced erythema of the face and of an erythrocyte count of 7,200,000 per cubic millimetre in such a patient during treatment.

During the past years the transformation of polycythæmia into myeloid leuchæmia has been commented upon. Harrop,[3] in a review of this disease, noted two such cases; Klumpp and Hertig[10] found only five of thirty-three patients recorded as suffering from erythræmia due to this disease; twelve suffered from leuchæmias, while in a few cases *post mortem* examination revealed the presence of both diseases and made differentiation impossible. In three long-standing cases of erythræmia Minot and Buckman[11] observed anæmia occurring coincidentally with further splenic enlargement and a leuchæmic blood picture; in one patient at autopsy the characteristic changes of myeloid leuchæmia in the spleen, liver and bone-marrow were found. Brieger and Forschbach[5] reported this metaplasia after deep X ray therapy, and the

following is a brief *résumé* of the notes of this patient.

In 1912 the hæmoglobin value was 150%, the erythrocytes numbered 14,000,000 and leucocytes numbered about 10,000 per cubic millimetre. Deep X ray therapy was given to the bones, and in 1920, after the patient had had further irradiation, the erythrocyte count was 4,600,000 and the leucocytes numbered 5,700 per cubic millimetre. Four months later, whilst the patient was under observation, the erythrocyte count dropped to 1,670,000, the hæmoglobin value dropped from 97·5% to 72·5%, and the leucocytes increased to 255,200 per cubic millimetre, with a differential count consisting mainly of myeloblasts and premyelocytes. Death occurred soon after. No autopsy was obtained, but "the true leuchæmic process was shown by the blood picture, by the appearance of extra-medullary foci in the fundi, liver and spleen, and also by an increased tendency to hæmorrhages and prolonged bleeding and coagulation times".

This paper records the termination of two cases of polycythæmia; one in a pronounced neutrophile leucocytosis and the other in myeloid leuchæmia. The histories are as follows:

Case I.

F.B., aged forty-seven years, was admitted to the Royal Melbourne Hospital on March 15, 1935. For four months she had complained of pain, and for three months of swelling in the left side of the abdomen. Although her appetite had been good, she had lost a small amount of weight; otherwise her history revealed no abnormal phenomena. On examination she appeared healthy; the systolic blood pressure was 154 and diastolic blood pressure was 90 millimetres of mercury, and the pulse rate was 90. The spleen was firm, painless and enlarged into the left iliac fossa; the liver had a regular, rounded edge and was palpable two fingers' breadth below the umbilical level. Routine examination revealed no other abnormality.

On March 18, 1935, the hæmoglobin value was 110% (15·1 grammes of hæmoglobin), the colour index was 0·72, erythrocytes numbered 7,620,000 and leucocytes numbered 41,600 per cubic millimetre inclusive of nucleated red cells (see Table I). The erythrocytes were fairly uniform in size, but very microcytic; a halometer reading showed an average diameter of 6·3 μ approximately. Most of the cells were hypochromic; a few showed polychromasia, and reticulocytes numbered about 4%. In counting, 200 leucocytes, 13 normoblasts, 18 macronormoblasts and 2 megaloblasts were observed. The leucocytes were neutrophile, with a pronounced shift to the left of the granular series, immature cells as far back as premyelocytes being present (Table II). It was difficult to obtain an accurate differential count because the blood was so viscous that thin films could not be obtained without many of the leucocytes being damaged. Platelets numbered 270,000 per cubic millimetre; many appeared much larger than normal. The erythrocytes formed 62% and the plasma formed only 38% of the total blood volume. The blood sedimentation rate was very slow and showed only a 0·5 millimetre fall at the end of two hours. There was no reaction to the Van den Bergh test, whilst the coagulation and bleeding times were within normal limits, the methods of Lee and White and of Duke respectively being used. The above blood findings were consistent with a diagnosis of *polycythæmia vera* in which the bone-marrow hyperplasia involved both the myeloblastic as well as the erythroblastic marrow.

Deep X ray therapy was commenced on March 27, 1935, and the progress of the patient's blood changes is shown in Table I. With periodic irradiation she was kept in fairly good health until three months prior to her readmission to hospital on August 20, 1936. During this time she suffered from a constant aching pain in the left side associated with shortness of breath, anorexia and a loss of two stone in weight. The patient looked emaciated and ill. The systolic blood pressure was 140 and the diastolic blood pressure was 70 millimetres of mercury, and the pulse rate was 100. The spleen extended into the left iliac fossa, whilst the liver reached the umbilical level. No glands were palpable. A blood count revealed a reduction in the erythrocytes and an increase in the leucocytes to 45,500 cells per cubic millimetre (Table I). Microscopic examination of the erythrocytes revealed considerable anisocytosis, poikilocytosis, polychromasia and hypochromia. In counting, 100 leucocytes, 21 normoblasts, 20 late erythroblasts and 4 early erythroblasts were seen. In a differential count of the leucocytes many were found to be of the primitive type and belonged to the myeloid series (Table II).

Examination of the fundi on September 1, 1936, revealed the characteristic leuchæmic type of hæmorrhages. The

TABLE I.
Blood Counts of a Patient with Polycythæmia.

Dates.		Hæmoglobin Value (Sahli).	Erythrocytes.	Leucocytes.	Colour Index.	Abdominal Findings.
Deep X Ray Therapy.	Blood Count.					
	18/3/35	110% (15·1 grammes)	7,620,000	41,600	0·72	Spleen and liver three and two fingers breadth below umbilical level.
March 27, 1935, and April 11, 1935, to anterior and posterior abdomen.	31/3/35	100% (14 grammes)	7,280,000	7,500	0·68	
June 4, 11 and 21, 1935, to right and left loins	7/8/35	118%	7,500,000	15,000	0·78	Spleen and liver three and four fingers' breadth below costal margins.
January 3 and January 31, 1936 ..	3/1/36	127%	8,770,000	24,700	0·72	Liver and spleen one and five fingers' breadth below costal margins.
March 27, 1936, and April 8, 1936 ..	27/3/36	95%	7,100,000	14,800	0·65	
	30/6/36	76%	4,870,000	13,550	0·78	
	11/8/36	49%	2,820,100	24,450	0·87	
	20/8/36	65%	3,440,000	45,500	0·94	
	31/8/36	54%	3,770,000	68,800	0·85	

TABLE II.
Differential Leucocyte Count.

Date.	Total Leucocytes.	Polymorphonuclear Cells.			Old Metamyelocytes.	Young Metamyelocytes.	Myelocytes.	Premyelocytes.	Myeloblasts.	Mitotic Figures.	Monocytes.	Lymphocytes.
		Neutrophile.	Eosinophile.	Basophile.								
18/3/35	41,600	33	3	1·5	37·5	6·0	1·5	1·0	0	0	2·0	14·5
20/8/36	45,500	9	1	0·5	18·5	7·5	10·5	20·0	15·0	0·5	0	17·5
31/8/36	68,800	9	1	0	12·5	7·5	13·5	21·5	23·5	0	1·5	10·0

patient gradually became weaker, and death occurred on September 9, 1936.

An autopsy performed twelve hours after death revealed pronounced mitral stenosis, as well as moderate atheroma of the aorta and of the renal, splenic and superior mesenteric vessels. The liver was grossly enlarged, with a smooth surface and slight capsular thickening. Its red substance, rather mottled with whitish areas, was found on microscopic examination to be widely infiltrated with myeloid tissue, and many nucleated cells were present. The enormously enlarged spleen, anchored by old fibrous adhesions to the diaphragm, was found to contain several small yellow infarcts at the upper pole. The splenic substance varied in appearance from a dark red to a uniform greyish-red colour; the Malpighian bodies could not be recognized, and the vessels were neither thickened nor thrombosed. Histological examination of the splenic substance revealed intense congestion, disappearance of the Malpighian bodies, dense infiltration with primitive myeloid cells, and the presence of many nucleated red cells. The rib marrow appeared pale greyish and rather fluid, whilst the femoral bone-marrow throughout the length of the shaft was a mixture of reddish and greyish solid tissue, the appearance being suggestive of great erythroblastic and myeloblastic proliferation. A smear from the bone-marrow revealed large numbers of primitive myeloid cells, of myeloblasts, of premyelocytes and of myelocytes, but only a few nucleated erythrocytes which were normoblasts. Microscopic examination revealed a hyperplasia of the myeloid elements and the presence of erythropoietic tissue in small islands, and examination of the kidney also revealed myeloid infiltration. A diagnosis of polycythæmia, myeloid leuchæmia and mitral stenosis was made.

When this patient was first seen in March, 1935, the findings were typical of *polycythæmia vera*. Great stimulation of the erythroblastic tissue was obvious, for many immature erythrocytes were present in the peripheral circulation; in addition there were similar activity of the leucopoietic tissues. Under deep X ray therapy the patient felt very well for a time, but, when her health gradually deteriorated, it was noted that a reduction in the erythrocyte count and a rise in the number of leucocytes had taken place, while the primitive myeloid cells had increased in number, and shortly before her death many myeloblasts were seen (Table II). Although no increase had occurred in the eosinophile and basophile cells (Table II), these findings indicated that the polycythæmia had been replaced by a terminal myeloid leuchæmia.

Case II.

W.M., a hairdresser, aged sixty-seven years, was first seen by a consultant on July 12, 1926, and gave the following history: Four months previously he had had a severe attack of pain in the right side which had doubled him up, had caused sweating and had lasted for over an hour. This pain recurred in a milder degree for the next three days, and was followed by malaise and inability to walk owing to swelling of the left ankle. There was no dyspnœa, but the patient had lost 4·5 kilograms (ten pounds) in weight during this period. His past history was as follows: A sore on the penis had developed in 1878; dental extractions in 1910 were followed by severe bleeding; an operation for hæmorrhoids in 1912 was associated with hæmorrhage; and after a hydrocele was tapped in 1922 a hæmatocele developed which required surgical treatment and orchidectomy. He had always had a tendency to free bleeding, but there was no familial history of hæmophilia. For many years he had been a heavy whisky drinker.

Examination of this healthy-looking old man, with ruddy ears, revealed a firm spleen which extended into the left iliac fossa. The liver edge was smooth and enlarged several fingers' breadths below the right costal margin. No glandular enlargements were noted. A slight trace of albumin was present in the urine, and on microscopic examination a few erythrocytes and leucocytes were seen. The hæmoglobin value was 125% (Sahli), the erythrocytes numbered 8,290,000 and the leucocytes numbered 18,800 per cubic millimetre. The colour index was 0·75. Apart from obvious anisocytosis with a slight tendency to macrocytosis and also some polychromasia, no abnormalities were observed in the erythrocytes and leucocytes.

The coagulation time measured by Dale and Laidlaw's method was two minutes five seconds; the bleeding time was prolonged to thirty minutes. On July 29, 1936, the patient was admitted to hospital for further investigation, and also to arrest the bleeding which followed a diagnostic puncture of the ear and which had lasted for thirty-six hours. The response of the blood to the Wassermann test confirmed the presence of syphilis. The blood urea was 29 milligrammes per 100 cubic centimetres of blood. A diagnosis of syphilitic splenomegaly with polycythæmia was made. A puzzling feature was the very prolonged bleeding time with normal platelets, an uncommon finding in polycythæmia. He was transferred to the out-patient department and was given a course of anti-syphilitic treatment, mainly in the form of five injections of "Novarsenobillon" and a small amount of potassium iodide.

TABLE III.
Blood Examination of a Patient with Polycythæmia.

Date.	Hæmoglobin (Sahli).	Erythrocytes per Cubic Millimetre.	Leucocytes per Cubic Millimetre.	Colour Index.	Platelets per Cubic Millimetre.	Appearance of the Film.
	%					
12/7/26	125	8,290,000	18,800	0·75		Anisocytosis with slight tendency to macrocytosis. Slight polychromasia.
19/7/26	119	7,880,000		0·75	480,000	
4/8/26	105	7,600,000	27,000	0·70		
11/10/26	88	5,328,000	26,800	0·82		Erythrocytes irregular in size and shape. Polychromasia. A few early polymorphonuclear cells, but no myelocytes.
22/11/26	105	7,360,000	16,200	0·71		
28/2/27	92	6,200,000	16,000	0·9		Film normal apart from some polychromasia.
30/5/27	90	?	19,400			
13/6/27		8,600,000				
17/11/27	104	7,000,000		0·74		
20/8/28	105	7,850,000		0·67		
5/3/29		6,700,000	18,000			Slight polychromasia and increase in average size of reds. Bleeding time, two minutes (normal, one to three minutes). Coagulation time, five minutes (normal, five to seven minutes). Slight polychromasia. One megaloblast seen.
14/8/29	85	7,280,000	18,300	0·6		
28/10/29	68	6,650,000	24,250	0·5		
2/12/29	70	6,770,000	24,100	0·5		
25/11/31	27	3,100,000	68,000	0·52		Considerable anisocytosis, definite hypochromia and slight poikilocytosis. No increase of reticulocytes. No nucleated forms seen. An absolute neutrophilia with a definite shift to the left.
11/12/31			43,200			Differential count as on 25/11/31.

TABLE IV.

Differential Count.

Date.	Total Leucocytes.	Polymorphonuclear Cells.			Old Metamyelocytes.	Young Metamyelocytes.	Myelocytes.	Premyelocytes.	Myeloblasts.	Monocytes.	Lymphocytes.
		Neutrophile.	Eosinophile.	Basophile.							
12/7/26	18,000	82·0	1·0	0	3·0	3·0	0	0	0	2·0	9·0
28/2/27	16,000	85·0	0	0	2·0	0	0	0	0	3·0	10·0
30/5/27	19,400	90·0	0	0	1·0	0	0	0	0	1·0	8·0
14/8/29	18,300	72·0	1·0	0	5·0	0	0	0	0	0	22·0
25/11/31	68,000	60·5	3·0	0·5	27·5	1·5	0	0	0	3·5	3·5
11/12/31	43,000	56·5	0·5	0	35·5	2·0	0·5	0	0	1·5	3·5

The latter drug was poorly tolerated and had to be discontinued.

The total blood volume after several days of hæmorrhage from the ear was estimated to be 73 cubic centimetres per kilogram of body weight, and suggested absence of plethora. The basal metabolic rate was found to be *plus* 40%, an elevation commonly noted in polycythæmia. During attendance at hospital his health was good, apart from occasional attacks of gout in the great toe and fingers, which were promptly relieved by colchicum and "Atophan". On May 7, 1929, the blood did not react to the Wassermann test. Readmitted to hospital as an in-patient on November 24, 1931, he stated that his health had been good till eight weeks previously, when generalized aches, fever, cough and sputum developed. On getting up after three weeks in bed he felt very weak and dyspnœic. The heart was enlarged to the right and left, and a loud systolic murmur was heard all over the præcordium. The spleen was enlarged one hand's breadth below the left costal margin and the liver was palpable. Swelling and bony thickening were present in most of the joints of the hands and grating in the metatarsophalangeal joints of the left hallux was also noticed.

A blood examination revealed a great decrease in both the hæmoglobin and erythrocyte levels and an increase in the leucocytes to 68,000 per cubic millimetre (Table III). In the differential count made on December 11, 1931, 93% of the cells were found to belong to the myeloid series, the majority being late metamyelocytes or polymorphonuclear cells (Table IV). The absence of increase in the eosinophile and basophile cells and of very primitive white cells ruled out a diagnosis of myeloid leuchæmia in this case. It is well known that under certain conditions and also in polycythæmia pronounced neutrophile leucocytosis with an increase in the number of immature cells, which at times even exceed 100,000 per cubic millimetre, may occur. In this case the polycythæmia in the later stage of the illness showed features characteristic of severe secondary anæmia with associated neutrophilia.

He was referred again to the out-patient department for another course of anti-syphilitic treatment, but he attended once only, early in 1932. His general condition gradually deteriorated and he died at home in June, 1932. Unfortunately during the latter part of his illness no blood counts were made and permission for an autopsy was not obtained.

Summary.

1. Erythræmia and myeloid leuchæmia are closely related from a pathological standpoint, and in some cases of erythræmia involvement of both the erythroblastic and the leucoblastic tissues occurs. Two cases are reported in which the illness commenced as polycythæmia, in one primary and in the other possibly secondary syphilis, and terminated as myeloid leuchæmia and secondary anæmia with pronounced neutrophilia respectively.

2. This transformation into myeloid leuchæmia is rare. In view of the increased number of leucocytes, an almost constant accompaniment of erythræmia, the term erythro-leuchæmia has been suggested. It is better to avoid this term and to describe the disease as either erythræmia or leuchæmia.

3. No satisfactory explanation for this occurrence has been given. In certain cases the condition has followed irradiation of the spleen and of the long bones, but this is possibly only an accidental happening. The solution appears to lie in the discovery of the ætiology of both erythræmia and leuchæmia.

4. As all grades of transition between polycythæmia and myeloid leuchæmia exist, differential leucocyte counts at intervals are necessary to determine whether an increase in the primitive myeloid cells is occurring—an event which may be associated with the development of leuchæmia.

Acknowledgements.

I am indebted to the medical staff of the Royal Melbourne Hospital for permission to record these case histories, to the pathological department for their frequent blood examinations, and to Dr. R. J. Wright-Smith for the use of his *post mortem* notes.

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FRACTURE OF THE PELVIS.[1]

By J. B. COLQUHOUN,
Melbourne.

FRACTURES generally, if one reads the signs of modern tendencies in surgery, are gradually gravitating to specialists in fracture work. Owing to the frequency of trauma in modern life, a new specialist is rapidly developing—the traumatic surgeon. With the high development of Röntgenology, with compulsory insurance and with the results of team work in the last war, it was inevitable that special services should be established to deal with the results of injury in this mechanical age.

Not, however, until Lorenz Böhler, of the Accident Hospital in Vienna, had persuaded insurance companies that it paid them to have their own hospital for the treatment of accidents did it dawn on our conservative profession that a fracture service was essential in every modern hospital. The insurance companies found that it was profitable business to have clients treated in well-equipped, well-organized clinics by people specially trained and specially interested in fractures. No one will dispute the fact that this development has been of advantage to the patient. I do not suggest that all fractures should be treated by traumatic surgeons; but they should be treated by those who are interested in mechanical problems and who have been trained to avail themselves of modern mechanical means, so that as perfect anatomical results as possible will be secured.

Fracture clinics have developed because of the frequent attitude of *laissez-faire* adopted by surgeons towards their fracture patients. In no other group of fractures has this been more apparent than in fractures of the pelvis.

Pelvic fractures, then, may be classified broadly into two main groups: (i) fractures which do not involve viscera, and (ii) fractures which involve the viscera. My observations are based chiefly on patients with this fracture admitted to the Royal Melbourne Hospital and to the Children's Hospital in Melbourne during the last five years, and I wish here to express my thanks to the staffs of both hospitals for permission to do this.

Historical.

In a volume of surgery by Lawrence Heister, of Helmstadt, published in 1743, we find the following:

> When the *os innomenatum* is broken, which seldom happens, it is readily discovered by the injury and symptoms in the neighbouring parts and is more particularly dangerous when the patient discharges a brown and bloody matter. In restoring this bone the patient must lie down on his sound side; the bone is to be replaced with the hands covered with compresses dipped in *Spiritum Vini* and bound up with the bandage spica. Afterwards bleeding with cooling and relaxing medicines must be used and a thin diet observed.

In fractures of the sacrum the same author advises:

> Or if he must needs sit at times let it be in a chair without a bottom to avoid displacing of the bone from touching the seat.

In the last 200 years much progress has been made owing to the introduction of asepsis and skeletal methods of traction, but in simple fractures little more is done than was done in the days of Heister.

Incidence.

During the last five years 139 patients who had sustained a fracture of the pelvis have been admitted to the Melbourne Hospital; of these nineteen died, a mortality of 13·75%. In the Children's Hospital during ten years twenty-three such patients were admitted, of whom three died, a mortality of 13%. The fractures occurred as frequently in girls as they did in boys, whereas at the Melbourne Hospital males outnumbered females by three to one. This fracture has become more frequent since the introduction of speedier motor cars during the last three years. In over 90% of cases fractures of the pelvis are due to the victims' being knocked down or run over by motor vehicles. In adults the fracture is due to severe trauma, which crushes the pelvic girdle; the motor car is easily the most common agent. Falling from a great height and being struck by heavy falling objects are common causes. In one case fracture of the superoposterior lip of the acetabulum was caused by a fall while the victim was playing cricket.

Classification.

No very satisfactory classification for fractures of the pelvis has been worked out, and I am not going to attempt to classify these fractures except in the general way which I have already indicated.

Owing to the severity of the injury most patients are brought to hospital suffering from profound shock, and frequently there is a history of unconsciousness. Practically every form of skeletal and visceral injury has been encountered in this series of cases, and because of this, treatment of the condition must be undertaken early in order to save life. In the early hours of treatment in many cases the fracture of the pelvis is of comparatively little importance. When shock is pronounced immediate treatment to relieve this must be instituted, and there is now a very strong tendency to employ blood transfusion at an early stage. Associated visceral or skeletal injuries must be treated *pari passu* with the shock.

When the associated injuries, which often are appalling, become contaminated with dirt from the street, prophylactic doses of antitetanic serum and of gas gangrene antiserum should never be withheld. In cases in which shock is a prominent feature, and in which there is associated skeletal injury, fracture of the pelvis may be overlooked. When examination is being made, however, of the victim of a "run-over" accident, or when severe crushing has occurred to the body, fracture of the pelvis should be suspected and the pelvis should be

[1] Read at the fifth session of the Australasian Medical Congress (British Medical Association), August, 1937.

examined by means of X rays while the patient is being taken from the casualty room to the ward or immediately afterwards by means of a portable X ray unit. As a rule, however, the history and the localization of pain in the region of the pelvis make the diagnosis of this fracture relatively easy. Inspection, palpation and rectal examination should be carried out as a routine measure, but a clear picture of the extent of damage to the pelvic girdle can be obtained only from good X ray films. In simple cases of fracture without associated fractures in other regions, and when there is no evidence of injury to the bladder, urethra or intestines, treatment for shock and some simple method of immobilization are all that is necessary. Such treatment was carried out 200 years ago, and in a review of records of cases in Melbourne I find that very little more is now done.

At the Children's Hospital only four cases of rupture of the urethra occurred in twenty-three cases of fracture of the pelvis. In one of these rupture of the bladder was associated, and in another rupture of the rectum. In three cases there was slight hæmaturia, but no lesion of the bladder or urethra was discovered; these patients recovered without complications. Of the patients who died at the Children's Hospital two were very severely injured and they died in the casualty room. As no *post mortem* examination was made I am unable to say to what extent there was internal injury. In following up the patients discharged from the Children's Hospital I found that healing of the fracture had occurred without gross deformity and with no disability whatsoever in the eight patients examined. In one case in which repeated attempts were made to repair a perineal fistula, the child presented himself with a suprapubic fistula through which all his urine was voided. This child volunteered the information that he could hold his urine under control for from three to four hours. At the Royal Melbourne Hospital nineteen patients died as a result of fractured pelvis and associated injuries; 90% of these died within a week of injury, and they all had suffered severe injury of other parts. Only one patient contracted bronchopneumonia, and two died from septicæmia associated with extensive lacerations and rupture of the urethra, while sixteen died from severe shock which could not be relieved by active treatment.

Treatment.

When fracture of the pelvis is suspected, the patient should be placed gently on a stretcher with a pillow under the knees to secure flexion of the hips and knees, and the knees should be bound together. In most cases X ray examination will reveal little or no displacement. For such patients a Bradford frame with a firm pelvic binder and pillow under the knees will be satisfactory. When displacement is present, as a rule gradual traction to reduce the displacement is sounder practice than manipulation on account of the danger of damage to the pelvic viscera by the latter method. When there is displacement of the acetabular portions of the pelvis, or concertina deformity and displacement, both legs should be encased in plaster of Paris from the groin to the toes, with a thick pad of felt to protect the skin on the medial aspect of the thigh. When the plaster is hard a block of wood is placed between the knees and a turn-buckle is applied at the level of the ankle; or, if this is not available, a Spanish windlass is used to approximate the ankles. In this way reduction may be obtained gradually and maintained with little danger of doing further damage to the pelvic contents until union of the fractures is secure.

This method may be tried when the head of the femur is displaced through the acetabulum into the pelvis. Since using this method, I believe it to be superior to the complicated method of Maxwell and Ruth. In the classical fracture of Malgaigne, in which the whole of one side of the pelvis may be displaced proximally, a canvas sling may be attached to a Balkan frame and counter weighted, and traction may be applied to a Braun splint (both hips and knees being flexed) to the inferior extremities. Counter-traction may be obtained by raising the foot of the bed one or two feet according to the displacement; in such cases a Bradford frame will not be required. In my experience the comfort obtained by the use of a double hip spica from the beginning when there is no displacement, and two to three weeks after reduction when displacement has occurred, makes me wonder why plaster is not more generally used. The spica is applied with the patient's knees flexed, and if a bar is placed to reinforce the plaster behind the knees quite a light cast may be employed. In this way nursing and general care of the patient are no longer a terror to the patient or a burden to the nursing staff.

Associated skeletal or visceral injuries may modify the method of treatment employed, but these may be so varied that this aspect of the problem cannot be dealt with here.

Treatment when Visceral Injury is Present.

Intraperitoneal rupture of the bladder is often missed, but if diagnosed it should be repaired and the abdomen should be drained. In extraperitoneal rupture the bladder should be repaired, the extravasated urine and blood should be removed and the region of the neck of the bladder should be drained.

Treatment in Injury to the Membranous Urethra Proximal to the Urogenital Diaphragm.

My own experience of rupture of the urethra is very limited, but from reviewing these cases and from the experience of others I have formed the opinion that definite rules should be formulated and that these should be strictly followed by those who have to do emergency surgery in the presence of such complications. When a rubber catheter cannot be introduced into the bladder it is extremely unwise to use rigid catheters or sounds. It is much better to perform perineotomy and drain the bladder, then to attempt to pass a catheter into the bladder and suture the urethra. Difficulty may be experienced in finding the proximal end of the

ruptured urethra, but if the bladder is compressed urine will be seen to escape. When the proximal urethra cannot be seen then the bladder must be opened and a catheter must be passed retrograde to the perineotomy wound. This procedure might well be adopted as the routine treatment in emergency work. Patients in whom rigid catheters have been used not too gently have been the ones who have given most trouble, and they usually finish with a perineal fistula. Suture of the urethra, like suture of cut nerves and tendons, should be left to those who have had special experience and training if even moderate results are to be expected. It is not evidence of incompetence, but evidence of strength of character, to pass these patients over when this is possible. In this way several surgeons would become experienced in dealing with this distressing condition which may have such far-reaching results. All of you would come to this conclusion in the interests of your patient if you carried out an investigation of a large series of patients treated in any large teaching hospital. After all, the patient's interests are the only factor that should be considered.

PERSONAL EXPERIENCE IN THE SURGERY OF THE COMMON BILE DUCT.[1]

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IN delivering this address and in choosing the subject I have, I have been guided by several considerations. When a subdivision is honoured by a meeting of the Branch, it is right that the subdivision should make some attempt to provide a paper. This, however, places the subdivision in a predicament, for conditions of practice being what they are, it is difficult, if not impossible, for us to read a paper of any real value. Sometimes, however, it is helpful to have a homely discussion, and it is with the purpose of stimulating this that I offer my own remarks.

The subject has been chosen, not only because it is of interest to surgeons, but particularly because it is apparently becoming the practice for more and more of us to perform our own operations on the gall-bladder, and I think it is wise that occasionally we should pause to consider the difficulties that may beset our path.

No attempt will be made to deal at all comprehensively with the various lesions which may affect the common bile duct, for numerous articles are available in many text-books and periodicals; rather shall I confine my remarks to my own cases. These fall under three headings: (*a*) obstruction of the common duct by carcinoma of the pancreas, (*b*) obstruction of the common duct by stones, (*c*) obstruction of the common duct from other causes.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 10, 1937.

Obstruction of the Common Bile Duct by Carcinoma of the Pancreas.

I have had four cases in the first group, the growth being in the head of the pancreas. All patients had the typical history of progressive jaundice with loss of weight and little or no pain. In two the gall-bladder was anastomosed to the stomach and in two to the jejunum. Three patients obtained relief for some months; the remaining patient, whose condition was advanced and whose operation was difficult, died after operation.

Obstruction of the Common Bile Duct by Stones.

The second group consists of 23 cases. Some of the main details are as follows.

Of the 23 patients, 19 were females and four were males. There was no jaundice or history of jaundice in nine cases, or 39%. Biliary colic was absent in seven cases, or 30%. Twenty-one patients were submitted to operation and three died; two were not operated on and both died.

Two patients were in the 20 to 30 age group; five were in the 30 to 40 age group; five were in the 40 to 50 group; six were in the 50 to 60 group, and five were in the 60 to 70 group.

Diagnosis.

The diagnosis of these cases may be worth discussing, for it does not yet seem to be realized fully that stones may lie in the common duct without ever causing jaundice or colic. Maingot[(1)] states that "some twenty-five per cent. of patients with stones in the common bile duct have never had jaundice, and some five per cent. do not even give a history of pain". In this small series 39% had not been jaundiced and 30% had had no severe pain. Three patients had had neither pain nor jaundice.

Three patients had, several years earlier, been diagnosed as suffering from catarrhal jaundice, and for this, if for no other reason, it would appear that such a diagnosis should be made with a certain amount of mental reservation.

An operative diagnosis may be equally difficult. Palpation of the ducts is generally accepted to be of little value, and there are many surgeons who state categorically that no operation on the gall-bladder is complete without exploration of the ducts. Maingot, who discusses this question with much wisdom, advises that the ducts should be explored in all cases except when the condition of the parts or of the patient renders this impossible or inadvisable. Personally I have not conformed to this standard, for I have been guided by what I have been informed was the practice of Sir Alexander MacCormick, that is, to explore the ducts when either the history of the patient or the appearance of the ducts raises any suspicion that stones may be present. Acting on this advice, I have many times explored the ducts fruitlessly and also I have on two occasions, to my knowledge, left stones behind. These stones would have been left also if I had been conforming to Maingot's standard. On

one occasion only has exploration of the ducts caused any trouble. These cases will be described later.

Operation Details.

In twenty of the twenty-one operation cases stones were removed by supraduodenal choledochostomy; the remaining stone was removed by the transduodenal route. A description of this latter case may be helpful to some. It was my first "solo" operation on the gall-bladder, and I had removed the gall-bladder before a stone was noted in the lower end of the common duct.

In trying to extract the stone I broke it and I was unable to pass any instrument through the duodenal papilla. I had been taught that in these circumstances stones would inevitably reform, so there was nothing for it but to open the duodenum and try to clear the duct from below. By this time, of course, the stone could not be felt, and I can assure those who have not tried it, that looking for the papilla amongst the folds of duodenal mucosa is an unenviable task. I could not find it, so a Hegar's dilator was passed as far as possible down the duct and then cut down on through the posterior wall of the duodenum. Some fragments of stone were removed and the cut edges of the duct were sutured to the opening in the posterior wall of the duodenum. I did not know then that this operation is dignified by the title of Kocher's transduodenal choledochostomy; but at any rate the patient made a good recovery.

Two operations were performed in the presence of severe jaundice and white bile in the ducts. Both patients were young women. In one the gall-bladder was removed as well as a solitary stone in the common duct; in the other a solitary stone was simply removed from the duct. The first patient recovered and the second died, though the operation was very easy and took a minimum of time. In another patient with severe jaundice the duct contained dark jelly-like bile as well as multiple stones. I should like to give the history of this patient in more detail, as it illustrates some of the difficulties that may be encountered.

Mrs. K., a stout woman of fifty-one, had had steadily increasing jaundice for one month with some, but not severe, pain. Her general condition was good. Two days later, while undergoing pre-operative treatment, she became very drowsy. Operation was performed on the following day with the intention of doing the bare minimum. The common duct was exposed with difficulty, as the first part of the duodenum was drawn well up to the *porta hepatis*. On the duct being opened a quantity of black jelly was extruded, but no fluid bile. Stones were impacted in the duct both above and below the opening, so the duct was cleared, four large and a number of small stones being removed. There was no leakage of clear bile throughout this performance. The gall-bladder was felt to contain a quantity of grit as well as two large stones. As the cystic duct was of wide bore, it was thought necessary to remove the gall-bladder so as to obviate the risk of the common duct being quickly reblocked. Death occurred eight hours after operation, and during this time not more than a staining of bile drained from the tube in the common duct.

Throughout the whole conduct of this case my hand was forced at every turn, and for my own instruction I should welcome frank criticism.

One case is noteworthy, in that thirteen very large stones were removed from the common duct while the gall-bladder contained none. This patient had never been jaundiced and had had no pain prior to the severe attack which brought her to hospital. Possibly this was an example of primary stone formation in the liver, to which attention has been drawn by Digby.[2]

Another patient had a fistula between the gall-bladder and duodenum as well as stones in the common duct. This patient had had a cholecystostomy performed fourteen years previously.

Several patients have had the common duct blocked by biliary sand or mud, and I have had the uneasy feeling that, as some of this *débris* is probably formed in the liver, the duct may become reblocked. To my knowledge this has not happened.

"Recurrent Stones."

I have had no case of true recurrent stone, but there have been four cases in which stones were left behind at a previous operation. I was the culprit on two of these occasions. The notes of these cases are as follow:

Mrs. B., aged forty-six years, had had cholecystectomy performed elsewhere. During the next three months she had repeated attacks of colic and mild jaundice. One large stone was then removed from the supraduodenal part of the common duct.

Mrs. D., aged thirty-six years, weighed fifteen stone. Cholecystectomy was performed for acute empyema of the gall-bladder. Much œdema of the tissues was present. During the next six months she had several attacks of colic but no jaundice. A stone, the size of a duck shot, was then removed from the ampulla of Vater.

Mrs. R., aged fifty-seven years had had dyspepsia and pain at the right costal margin for many years. She had no colic or jaundice. Operation revealed a hopelessly fused mass, consisting of liver, omentum and bowel. After dissection a hard eminence was felt within the mass. Aspiration of bile showed this to be the fundus of the gall-bladder. This was opened, its wall being over one centimetre thick and extensively calcified. Numerous calculi were extracted, the last one being huge. Very little bile drained, and ten days later the patient became jaundiced and the motions clay-coloured. Further operation was felt to be hopeless, but was advised. It was refused and the patient died three months later.

Mrs. A., aged thirty-three years, had had cholecystectomy performed elsewhere. She had never been jaundiced. Three months after operation she had an attack of colic and jaundice. During the next five months the jaundice persisted, with some variation in depth, and there were several bouts of colic. She lost two stone in weight. When first seen, she was extremely jaundiced, but the general condition appeared good. The liver was grossly enlarged and there was some ascites. As Wright[(3)] had just published the notes of a case in which pre-operative oral administration of bile had appeared to be most beneficial, this patient also was given salol-coated bile tablets. Vomiting ensued two days later. The tablets were stopped and the vomiting ceased. After two days the tablets were recommenced. Vomiting immediately recurred and was quickly followed by complete suppression of urine, multiple gross subcutaneous hæmorrhages and death.

This last case is particularly interesting. Wright emphasized that the bile tablets must be coated with salol so that they should not be dissolved in the stomach and irritate it. In this case it seems reasonably certain that it was the bile tablets that precipitated the crisis.

Mortality.

The mortality of the series has already been stated. To recapitulate, the two patients who were not operated on, and three out of twenty-one who were submitted to operation, died. All the fatal cases, except one, have already been described. In the remaining case death occurred suddenly, three weeks after operation, from a cardiac infarct.

Obstruction of the Common Bile Duct from Other Causes.

The third group consists of eight cases. Obstruction was caused by hydatid *débris* in one case. In no case was obstruction caused by chronic pancreatitis. In seven cases the obstruction was from an unspecified cause.

The patient whose common duct was obstructed by hydatid *débris* made a good recovery. The cyst was not seen.

Chronic pancreatitis has been mentioned for purposes of discussion. My own opinion is that it is very seldom, if ever, a cause of biliary obstruction, for I have never seen, or at any rate never recognized, such a cause. Also my friend Dr. Wright-Smith tells me that in a series of some six thousand autopsies he has not encountered a true case of this nature.

The remaining seven cases I propose to set out in detail.

Mr. W., aged sixty-nine years, gave one month's history of moderate pain, progressive jaundice and loss of weight. He was extremely jaundiced. The gall-bladder was found to be tense with white bile, but it contained no stones. The common duct also contained white bile but no stone. A probe was passed through the duodenal papilla with much difficulty. The gall-bladder and common duct were drained. The patient made a good recovery and has remained well for over a year.

Mrs. H., aged sixty years, gave a recent history of dyspepsia and frequent vomiting. She had one non-faceted stone in the gall-bladder. The common duct was very large. A probe was passed with slight difficulty into the duodenum. Cholecystectomy was performed. The patient has improved, but still has some dyspepsia.

Mrs. G., aged fifty-nine years, gave a long history of biliary colic and slight jaundice. Numerous stones were present in the gall-bladder. The common duct was very large. A probe was passed to the duodenum with some difficulty. Cholecystectomy and choledochostomy were performed. Very copious post-operative drainage of bile resulted. The patient has remained well for two years.

Mrs. S., aged fifty years, had had dyspepsia and moderate pain for one year. She was never jaundiced. Her small gall-bladder contained many stones. The common duct was very large. A probe was passed to the duodenum with some difficulty. Cholecystectomy was performed. The patient has remained well for a year.

Mrs. T., aged fifty-three years, had had numerous attacks of biliary colic for some years with chronic jaundice. The gall-bladder was atrophic and contained four stones. The common duct was very large. A probe was passed to the duodenum with some difficulty. Cholecystectomy was performed. The patient has remained well for three years.

Mrs. H., aged sixty-one years, had splenectomy performed elsewhere for hydatid disease of the spleen thirteen years earlier. She had severe biliary colic without jaundice for ten years. Extremely dense adhesions were present at the lower margin of the liver. After much dissection a complete gall-bladder, less than two centimetres long, was exposed. It contained no stones. The common duct was very large. A probe was passed to the duodenum with moderate ease. Cholecystectomy was performed. She has remained well for nine months.

Miss W., aged forty years, had had severe biliary colic with some jaundice for several years. She had an atrophic gall-bladder with many stones. The common duct was moderately enlarged. A probe was passed to the duodenum with some difficulty. Cholecystectomy was performed. A huge abscess formed below the transverse mesocolon and medial to the ascending colon; and later a duodenal fistula formed. She made a good recovery and has remained well for over a year.

In the first of the cases described under this heading it seems probable that the obstruction of the common duct had been due to a stone which had ulcerated through the duodenal papilla and left a scar which had contracted; or it may be that a stone was pushed into the duodenum by the probe. It may be argued that one of these conditions was also present in the remaining six cases, but in these latter cases such an argument seems far-fetched. Nor was there any evidence that the dilatation of the common duct was due to chronic pancreatitis. My own belief is that in the latter cases the dilatation was due to spasm of the sphincter of Oddi, or rather to achalasia of this muscle, and it is interesting to speculate whether this non-relaxation was induced by the diseased condition of the gall-bladder or *vice versa*. It would not be profitable to debate this question here, but it is desired to stress the fact that these cases resemble exactly, both on clinical and anatomical grounds, those in which the common duct is blocked by stones.

In British literature there is little reference to this condition, though Hurst[4] has for some years been drawing attention to its occurrence after biliary operations, and the non-operative treatment of cholecystitis has for long aimed at producing relaxation of the sphincter of Oddi and a free flow of bile. In foreign literature, under the name of biliary dyskinesia or dyssynergia, this condition is receiving much attention, and apparently there is a tendency to ascribe too many clinical states as due to it, for recently Mentzer[5] has thought it necessary to utter a warning against too lightly making such a non-operative diagnosis.

Before leaving this subject I should like to give the history of what may be a true case of biliary dyskinesia.

Mr. R., aged twenty-two years, had suffered from severe asthma for years. For the last three years he had been subject to increasingly frequent attacks of severe pain in the upper part of the abdomen, followed sometimes by slight jaundice. Appendicectomy had been performed in one of the attacks. A cholecystogram had not revealed any abnormality of the gall-bladder. He was first seen by me in an attack of acute biliary colic. At operation two days later the gall-bladder appeared normal and the common duct was not dilated. The duct, when opened, contained clear bile, and a probe was passed into the duodenum with moderate difficulty. The sphincter of Oddi was dilated, the gall-bladder was removed and the common duct was closed. The gall-bladder was quite normal to macroscopic examination. In the nine months since operation there have been no abdominal symptoms of any kind, nor has any attempt been made to prevent them. The patient has undergone desensitization treatment for asthma with a fair amount of success.

Conclusion.

This disjointed description of cases is a complete account of one person's experience in the surgery of the common bile duct. In my own gall-bladder cases the incidence of lesions of the common duct has at times been extraordinarily high, and not infrequently such lesions have been found when not expected.

Acknowledgement.

I should like to take this opportunity of acknowledging my debt to the surgeons of the Royal Melbourne Hospital who were patient enough to instruct me in this branch of surgery.

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BLOOD UREA CLEARANCE TESTS.

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Honorary Physician to Out-Patients, Royal Melbourne Hospital.

THERE is no doubt that the blood urea clearance test is being used more extensively than previously, and that it is considered in other countries to be a very useful test of renal function. However, there is a wide variation in the functional efficiency of even healthy kidneys at different times, and there may be a very great difference between the values that are obtained from two hourly specimens of urine. Bruger and Mosenthal[1] found the value to be twice as high in one hour as in the other; they found a value of only 58% in a normal subject, and Fishberg[2] observed it to be below 50% as a result of cardiac failure, so that there appears to be a need for some improvement in the original technique.

It is unnecessary to comment in detail on Van Slyke's technique, which has already been discussed in this journal.[3] It is sufficient to state that routine tests by this method did not appear to possess any material advantage over the estimation of blood urea, urea concentration, urea elimination, and urea concentration factor, when they were applied to determine renal function in any isolated case. It must be recognized that the absolute value of the renal function in any normal person cannot be determined by this or by any other test; but, as Bruger and Mosenthal[1] stress, as the urea clearance diminishes with impaired renal efficiency, it shows fewer variations and becomes more and more fixed. Clearance values which are repeatedly and persistently low indicate fixation of renal effort, which is characteristic of impairment.

It is therefore in the early stages of impairment that there is most difficulty in interpreting this test; but it must be remembered that subnormal values obtained during the last two months of pregnancy must be interpreted with extreme caution, particularly in the absence of other evidence of renal dysfunction.[4]

Qualitative tests of renal function cannot be displaced by the use of more elaborate tests, and too much stress cannot be placed on the extreme value of thorough clinical examination of the urine, of repeated microscopic examination, of Volhard's dilution and concentration test as modified by Fishberg,[2] and of Rabinowich's test for the general practitioner.[5] On the other hand, it cannot be denied that any move in the direction of exact quantitative estimation of renal efficiency is of value.

Fowweather[6] has suggested a modification of the test, which has the effect of improving the standardization of conditions for the blood urea clearance by the administration of urea, and a series of patients at the Royal Melbourne Hospital were submitted to the blood urea clearance test before and after the administration of fifteen grammes of urea.

The procedure which was adopted was as follows:

8 a.m.: Light breakfast; no coffee.
(i) 9 a.m.: Bladder emptied; urine discarded; time noted.
(ii) 10 a.m.: (*a*) Bladder emptied; exact time noted.
(*b*) Blood taken for urea estimation.
(*c*) Urea meal given.
(iii) 11 a.m.: Bladder emptied; exact time noted.
(iv) Blood taken for urea estimation.
(v) 12 noon: Bladder emptied; exact time noted.
(vi) 1 p.m.: Bladder emptied; exact time noted.

The blood urea clearance before the administration of urea was estimated from (ii) (*a*) and (ii) (*b*); the blood urea clearance after the administration of urea was calculated from (iv) and (v); the urea concentration and the urea elimination could be determined from (iii), (v) and (vi).

Details of the results of the investigation will be published elsewhere,[7] but some of the findings may be summarized and commented upon with advantage.

In eleven normal students it was found that the average rise in blood urea in one hour was 11·7 milligrammes per 100 cubic centimetres, with the extremes at 2·0 and 20·0 milligrammes. King[8] found an elevation of 15·0 milligrammes with an average rise of 10·1 milligrammes, and he found also that there was least variation in the level during the second hour. He quoted Rabinowich, who found an average rise of 9·5 milligrammes and maintained that the maximal rise was proportional to the amount of urea given; but the deviation was found to vary even in the same person. Fowweather[6] found in thirty-three normal students an average rise of 12·8 milligrammes with the extremes at 8·0 milligrammes and 16·8 milligrammes, the increase varying inversely as the weight of the individual; the level of the urea during the second hour varied from the average by less than 2·5 milli-

grammes in 76%, by 2·5 to 5·0 milligrammes in 12%, and by more than 5·0 milligrammes in only 9%.

From these findings it can be seen that in the normal person there will be a maximal rise of 15 to 20 milligrammes per 100 cubic centimetres in the blood urea after the administration of 15 grammes of urea, and that blood taken during this hour will be a fair sample.

The level of the blood urea in renal failure depends not only on the severity and duration of the latter, but also on the fluid intake, on the amount of protein in the diet, on the rate of katabolism of protein in the body, and probably on the functional condition of the liver. Circulatory failure will aggravate the retention of urea in renal failure, while improvement in the circulation will cause the level to fall. Pre-renal deviation, or azotæmia, and pre-renal oliguria are well-known causes of a raised blood urea. It is therefore evident that estimation of the fasting blood urea can have no very real advantage over the estimation of the blood urea after the urea meal. In actual cases it appeared to me that the latter was of much greater value, because a deviation from the normal was more evident in many instances. The rise of blood urea was considerably more than 20 milligrammes in 100 cubic centimetres when there was evidence of renal disease; and in some cases despite a variation of the fasting blood urea which would suggest improvement, the level after the urea meal tended to be remarkably constant. The urea meal to some extent obviates the effects of extrarenal factors.

The blood urea clearance values obtained after the urea meal were within a decidedly narrower range than those obtained before it in Fowweather's series of normals, and similar results were found in the Melbourne students, so that there is evidently less likelihood of normal patients giving subnormal values by this method (Figures I and II).

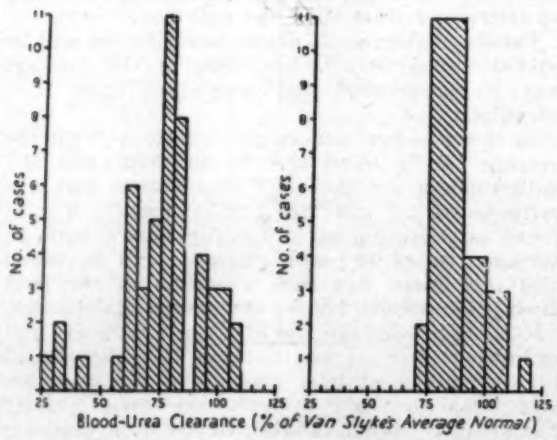

FIG. 1.—Before urea. FIG. 2.—After urea.

From Fowweather in *The Quarterly Journal of Medicine.*

In patients suffering from disease of the kidneys it was found that the blood urea clearance test after the urea meal appeared to give a more accurate indication of the degree of renal efficiency and to be more in keeping with clinical findings than the other tests; and in two instances in which autopsy showed definite chronic nephritis although the clearance values were normal, the other biochemical tests failed to disclose the presence of renal disease.

The conclusions arrived at were that the blood urea clearance test after the urea meal was of definite value, but that the procedure outlined above was rather too exacting for both the patient and the nursing staff, and was liable to result in inaccuracy. Accordingly, a slightly different technique has now been adopted.

The rate of urea excretion has been shown to be increased by the administration of milk, caffeine and glutamic acid by mouth, and decreased by exercise, pituitrin and large amounts of adrenaline; and these changes occur independently of changes in the blood urea concentration.[9] Therefore, it is desirable that the patient should have no breakfast on the morning of the test, and that he should have little or no exercise.

The blood is taken late in the second hour to allow of adequate absorption of the urea and in case of a delayed rise.

The recommended procedure is as follows:

No breakfast is to be taken on the morning of the test, but a glass of water may be taken at about 7 a.m. if particularly desired.

9 a.m.: Bladder to be emptied; the exact time to be noted to the nearest minute (for example, 9.1 a.m.). The urea meal to be given (15 grammes of urea in 100 cubic centimetres of water).

10 a.m.: Bladder to be emptied; the exact time to be noted (for example, 10.3 a.m.).

10.45 a.m.: Blood to be taken for urea estimation.

11 a.m.: Bladder to be emptied; the exact time to be noted (for example, 11.2 a.m.).

12 noon: Bladder to be emptied; the exact time to be noted (for example, 12 noon).

1 p.m.: Bladder to be emptied; time to be noted.

All of the urine passed must be sent to the laboratory; complete emptying of the bladder is essential.

Each specimen must be labelled with the name of the patient and with the exact time at which it was passed or taken.

All measurements will be made at the laboratory.

The urea clearance is determined from the blood urea and the third urine specimen, of which we can be sure of the exact volume, the exact time interval to the nearest minute, and the exact urea concentration. The urine specimens can be used for estimation of the urea concentration and urea elimination if desired, and collection of them has the effect of training the patient for the crucial specimen, so that the time will be exact and the bladder will be completely emptied.

Calculation of the Blood Urea Clearance.

It will be remembered that Austin *et alii*[10] found that, up to a certain point, an increase in

the volume of urine passed resulted in an increase in the ratio of the rate of urea excretion to the blood urea nitrogen. This increase was found to be approximately proportional to the square root of the urine volume excretion. As the rate of the volume excretion increased, a limit was reached beyond which increase of the rate had no further effect in augmenting the ratio. This they called the augmentation limit, the exact location of which in any individual is difficult. They found that it varied between 1·73 cubic centimetres and 4·16 cubic centimetres per minute, while Bruger and Mosenthal[1] found it to be between 1·67 and 2·55 cubic centimetres per minute; but 2·0 cubic centimetres per minute is considered to be the average augmentation limit.

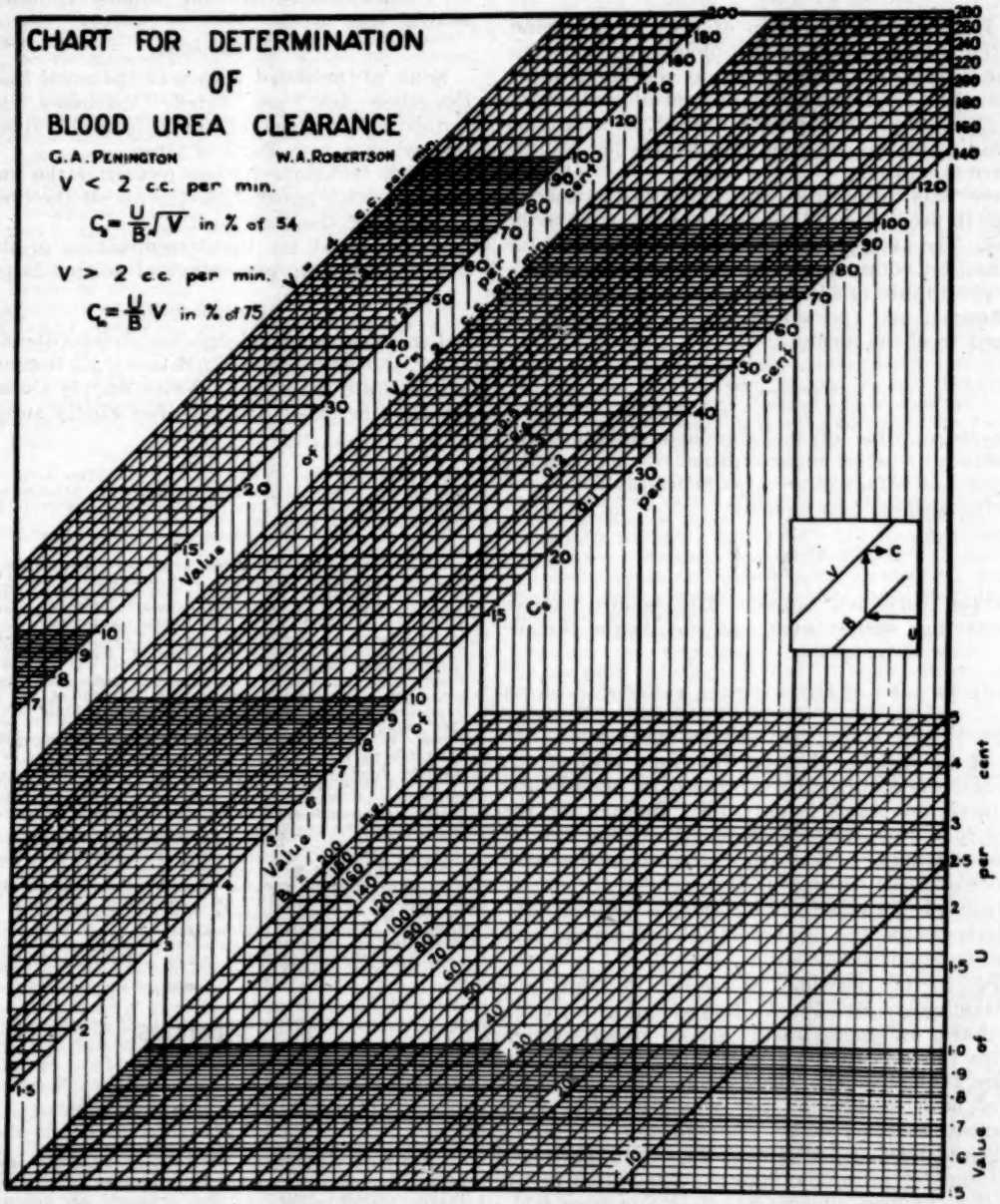

FIGURE III.

To determine the blood urea clearance in percentage of normal: find the value of *U* (urea concentration in percentage) and trace horizontally to the left to intersect the value of *B* (blood urea in milligrammes), thence vertically to the value of *V* (volume of urine in cubic centimetres per minute), and finally horizontally to the right to read the result.

Above the augmentation limit the blood urea clearance is called the maximum clearance or C_m, and the formula used is:

$$C_m = \frac{UV}{B}$$

when U is the concentration of urea in the urine, B is the concentration in the blood, and V is the volume output of urine in cubic centimetres per minute. The result may be expressed in cubic centimetres per minute or in percentages of the maximum clearance, which is 75 cubic centimetres per minute for normal adults.

Below this limit the clearance varies on an average in proportion to the square root of the urine volume. The average normal excretion of urine (or the standard volume) for adults is one cubic centimetre per minute (1,440 cubic centimetres in twenty-four hours), and the standard clearance or C_s is obtained from the formula:

$$C_s = \frac{U}{B}\sqrt{V}$$

The average value of the standard clearance for normal adults is 54 cubic centimetres per minute. Correction of V for surface area is desirable, and the following formula may be used:

$$V_c = \frac{1{\cdot}73}{S.A.}$$

V_c is the corrected volume, 1·73 square metres is the average surface area, and $S.A.$ is the surface area.

It will be noted that for a given value of $\frac{U}{B}$ and a volume of two cubic centimetres per minute, the value of C_m will be $\sqrt{2}$, or 1·414 times that of C_s; but when the results are expressed in percentages of normal, the result is only 1·8% greater for the former than it is for the latter. It is therefore an obvious advantage, and very desirable, that the results should be expressed in percentages.

Values for the blood urea clearance of over 65% in elderly people and of over 70% in others are considered to be normal.[6]

Since it is possible to solve the formulæ logarithmically, Mr. W. A. Robertson prepared a line chart on logarithmic graph paper, which obviates the necessity for arithmetical calculation (Figure III). The chart is used for the determination of both maximum and standard blood urea clearances, and the results are read directly in percentages of normal. It will be seen that there is a wide range for all of the factors. The extent of the range for the urea concentration (U) is from 0·5% to 5·0%, for the blood urea (B) is from 10 to 200 milligrammes, for the volume (V) is from 0·1 to 4·0 cubic centimetres, and for the percentages is from 1·5 to 280. The method of using the chart is indicated on it: commence with the urea concentration, read across to the blood urea, thence to urine volume, and finally back to the percentage. It must be remembered that the intervals between the lines must be divided logarithmically when intermediate values are being used. After a little practice it will be found that a high degree of accuracy is obtained with the use of the chart, and it can be recommended with the utmost confidence for routine use.

Summary.

Some of the shortcomings of the usual blood urea clearance test are briefly discussed, and an indication is given of the enhanced value of the test when a urea meal is given.

A new technique for the test after the urea meal is given, with reasons for the use of the blood urea values after the urea meal.

A line chart for the determination of the blood urea clearance in percentage of normal is provided.

Acknowledgement.

I wish to acknowledge my indebtedness to Mr. W. A. Robertson, M.C.E., M.Inst.C.E., Commissioner of the State Rivers and Water Supply Commission, for the line chart which he has kindly supplied.

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Reviews.

ORTHOPÆDIC SURGERY.

"RECENT ADVANCES IN ORTHOPÆDIC SURGERY" as the title of Burns and Ellis's book is somewhat of a misnomer, as their excellent little volume presents an account not so much of recent advances as of current thought in Great Britain on orthopædic problems.[1]

Assuming some knowledge by their readers of standard orthopædic practice, the authors make no claim to have produced a text-book. They are thus able, by omitting long descriptions of accepted facts and procedures, to

[1] "Recent Advances in Orthopædic Surgery", by B. H. Burns, B.A., B.Ch., F.R.C.S., and V. H. Ellis, M.A., B.Ch., F.R.C.S.; 1937. London: J. and A. Churchill Limited. Demy 8vo, pp. 304, with 108 illustrations. Price: 15s. net.

describe methods and conclusions in a manner which makes reading easy and interesting to the surgeon interested in the subject.

In a book of only 296 pages, including a good index, the range covered is very wide, and the full bibliography at the end of each chapter makes it easy for the reader to go more deeply into any phase of the subject.

The earlier chapters on bone, dealing with growth, transplants, chemistry and classification of tumours, are too brief to be more than outlines of the subject and could with advantage have been extended.

Since the object of the authors is, in spite of the title, to present accepted facts rather than the latest theories, there will be found very little to arouse adverse criticism in their expressed opinions.

A very valuable chapter in the book is that on painful shoulders, although the work quoted, mostly following Codman, dates back to 1911. The conditions causing pain in this region are perhaps less appreciated than those causing pain in any joint, and injuries to the supraspinatus tendon must certainly rank amongst the most common causes of disability.

The description of Pott's disease with its paraplegia is also particularly interesting to Australian surgeons, many of whom rarely have sufficient experience from their own cases to judge the value of operative procedures.

Altogether the volume serves a most useful purpose in presenting current views in tablet form; and yet is certainly not merely a synopsis, as it makes easy and pleasant reading.

THE AUTOBIOGRAPHY OF AN ITALIAN SURGEON.

IT is scarcely too much to say that no one sees more of the drama of life than the surgeon; and this is particularly true of the surgeon attached to a large general hospital. Professor Andrea Majocchi, of Milan, in his "Life and Death" has written an account of his own career by which this statement is fully justified.[1] He has left out nothing. Beginning with the tragic death of his father from an infection caught at an autopsy, he tells of the difficulties associated with his education, of his fortunate winning of a bursary offered by the *Congregazione di Carita* that made it possible for him to go to college and to undertake medicine as a career, and finally, of his almost religious choice of surgery as his life's work. Before it was possible for him to specialize, however, he spent an unprofitable time (financially speaking) in general practice, and then by good fortune he became attached to the Obstetrical Station. All kinds of appalling experiences in the poorest quarters of Milan fell to his lot while he held this position, and he tells of them in so simple a manner that the dramatic effect is heightened. He determined to undertake general surgery rather than obstetrics, and accordingly, having been successful in winning a scholarship, he travelled to Switzerland and to America to learn what was to be learned from those countries. It is not surprising that he should have found American cities unbeautiful and American "mass-production" methods distasteful, ardent Italian as he was, although he willingly admitted the almost superhuman efficiency of the American hospitals.

> The Latin temperament rebels against this regimentation of human labour, this restriction of personal choice. It is the great universal geniuses, men like Leonardo, Michelangelo, Pico della Mirandola, and in medicine, the men of broad vision, the inspired clinician, the all-round surgical master, men of wide culture and varied activities, who command our ungrudging admiration.
>
> Americans and their enthusiastic pupils will not agree with me, but my Latin temperament will always incline me heavily to the belief that the rich and fruitful intuitions of the "universal" mind will always prove of greater service to humanity than the slick ingenuity of the single-track mind.

After his return from America, Majocchi spent some time in Berlin preparing a thesis on renal hæmaturia, and even then (1909) he and his fellow students ". . . exchanged glances and thought of the tragic conflict that was impending". Five years later he was to take part in the conflict, for he was attached to the *Città di Milano*, the first front-line temporary surgical hospital, from the moment its organization was complete, and he remained on active service until the end of the war. It is strange to read an account of the war in which Italy and Germany are treated as the combatant nations, and to learn that the victory was to Italy; the "Allies" are mentioned once or twice, no more.

Majocchi had to contend with professional jealousy after his period of foreign travel; he was more or less condemned because he was not university trained. However, these trials were not long-lived, and he was appointed consulting surgeon to the new obstetrical clinic, the Queen Helena's Home, after which his ability assured him success.

Not the least interesting chapter of the book is that devoted to the description of the great Italian pilgrimage to Lourdes, to which the author acted as physician. Another section of great interest is that dealing with post-war Italy and with the rise of the Fascists.

In no part of his autobiography does the author obtrude; rather his personality pervades the whole unostentatiously, as is proper. We see him as a man of extreme earnestness and of culture—as a man who takes his surgical responsibilities very seriously. To the non-medical reader his book may be alarming in parts, since he has written absolutely fearlessly of all the trials and difficulties that may beset a surgeon, not omitting dreaded "unavoidable accidents" such as post-operative embolism; but both medical and non-medical readers will find the book intensely interesting.

The translation has, on the whole, been well done, although the translator's unfamiliarity with medical terminology is to be regretted; but the author's pleasant philosophy has in no sense been interfered with by the transition from his native tongue to another.

SURGICAL PATHOLOGY OF THE THYREOID GLAND.

PATHOLOGISTS will probably not agree with all the conclusions that Hertzler has arrived at in his latest monograph on "Surgical Pathology of the Thyroid Gland", but we feel sure that they will appreciate his work on the subject.[1] On the other hand, practising surgeons will welcome this book. It is the eighth monograph in a series of ten on surgical pathology. Hertzler has at least attempted to give a complete picture from the surgeon's point of view of the pathology of this particular gland, and we believe that this is the first time that such a comprehensive outlook on the subject has been presented in a single publication.

The author is in a unique position, for he has been working for some forty years in a clinic situated in a country centre and he has in consequence been able to observe a large number of his patients from the beginning to the end of their diseases. He is unorthodox in many of his views, but this is not a disadvantage, for it indicates a man of original outlook. Hertzler was one of the first to advocate the total removal of the thyreoid gland in certain chronic diseases of the heart, and perhaps the first to recommend this radical procedure in many other recognized abnormal states of the gland—in so-called multiple adenomata, in atypical toxic goitre and in cases of myxœdema due to dysfunction of the gland

[1] "Life and Death: The Autobiography of a Surgeon", by Andrea Majocchi, Professor of Surgery, Milan; translated by H. J. Stenning. London: George Allen and Unwin Limited; 1937, pp. 343. Price: 10s. 6d.

[1] "Hertzler's Monographs on Surgical Pathology: Surgical Pathology of the Thyroid Gland", by A. E. Hertzler, M.D.; 1936. Philadelphia: J. B. Lippincott Company. Medium 8vo, pp. 316, with 238 illustrations.

previously described in his book on "Diseases of the Thyroid Gland", which was reviewed in this journal in the issue of January 18, 1936. Although he states that the use of a classification is only for beginners and for descriptive purposes, he adopts that recommended by the American Society for the Study of Goitre, but has added another entity, namely, cardiotoxic goitre. He shows that dysfunction of the gland is made evident not only by the microscopic appearances which are commonly described, but also by the minute changes in the cells themselves and in their staining properties, and also in the staining reactions of the colloid material. This is a reasonable outlook and one that demands the serious consideration of both pathologists and surgeons. One of the many lessons to be learned is that dysfunction of the gland is more harmful and needs more energetic treatment than hypersecretion and hyposecretion. Hertzler, in commenting on myxœdema, states that it is untenable to him that the myxœdematous state is one merely of diminished or absent thyreoid function, even though one finds cases which appear to be just that. On reading this chapter one is influenced by his teaching that the correct line of treatment in these cases is total removal of the gland as a preliminary measure rather than treatment by gland extract. Most surgeons of experience can call to mind instances in which removal of the gland or even part of the gland in patients showing signs of myxœdema has been followed by improvement, whereas treatment by gland extract has not produced satisfactory results. That hypersecretion and hyposecretion of the thyreoid gland are frequently associated with dysfunction of the gland is obvious to surgeons of experience and Hertzler rightly emphasizes this association.

The monograph, as is usual in his publications, is full of Hertzlerisms, which to some readers at least will make for more entertaining reading and at the same time emphasize his point of view. Surgeons may not agree with all that Hertzler writes and may not be so bold in their surgical procedure, but they must admit that, in the author's mind at any rate, the subject presents a complete picture and that there is little or no confusion.

Atypical toxic goitre, fœtal adenomata, myxœdema, tumours of the gland and thyreoiditis, together with many pages on adolescent goitre, which subjects have been scantily treated in the past in most text-books, have been dealt with thoroughly, and in many places show evidence of most painstaking work and original thought.

As is expected in publications by this author, the illustrations are excellent, and both he and the publishers are to be congratulated on the continuation of their high standard.

The work contains 279 pages, with a preface of 13 pages and an index of 18 pages. There are 238 illustrations, many of which show double pictures. We recommend this volume to the serious consideration of clinicians, pathologists and surgeons alike, for they will all find within its covers much that is stimulating and encouraging, and much from which knowledge can be acquired. Hertzler holds that in goitre, as in all other diseases, there must be a relationship between the pathology and the clinical manifestation; his book is an effort to demonstrate this relationship.

POST MORTEM EXAMINATIONS.

"AUTOPSY DIAGNOSIS AND TECHNIQUE", by Professor Otto Saphir, of Chicago, has been written to instruct the student, the interne and the "occasional" pathologist how to perform an autopsy and how to make a diagnosis from the findings.[1]

To avoid confusion the author describes only one method of autopsy technique—the one he has himself used for fifteen years and found advantageous. The body is opened by a Y-shaped cut, the limbs of the Y starting near the axillæ and meeting at the xiphoid, the stem of the Y passing down the mid-line to the pubis. All the organs from tongue to rectum are taken out *en masse* and dissected on the table. After the directions for dissecting an organ there follows a summary of the lesions to be looked for in it. The author wisely emphasizes the necessity of a routine order in examining the organs; otherwise important, though perhaps not obvious, lesions may be overlooked.

The sixty-five illustrations are mostly diagrams showing points in technique. There are hardly any illustrations of diseased organs, the author explaining that it was not his intention to write a text-book of pathological anatomy.

The book contains a great deal of information well arranged and succinctly expressed. A most useful feature is the series of tables on differential diagnosis showing for each organ the macroscopic points of distinction of its various diseases. The sixteen tables include outlines of kidney, liver, spleen, lung, thyreoid and intestinal diseases, the varieties of endocarditis, the causes of jaundice, the types of ascitic and pleural fluids *et cetera*.

Altogether the author's purpose is well carried out, and an autopsy guided by him will be thoroughly done.

[1] "Autopsy Diagnosis and Technique: A Manual for Medical Students, Practitioners, Pathologists and Coroners' Physicians", by O. Saphir, M.D., with a foreword by L. Hektoen, M.D.; 1937. New York: Paul B. Hoeber Incorporated; Australia: Angus and Robertson Limited. Crown 8vo, pp. 362, with 65 illustrations. Price: 30s. net.

A MANUAL OF ANATOMY.

MANY readers of this journal will be interested in the sixth edition of "Buchanan's Manual of Anatomy",[1] as the editor is Professor J. E. Frazer, of the University of London, the distinguished anatomist and author of "Anatomy of the Human Skeleton".

This manual of anatomy approximates in size to that of the well-known text-books of anatomy. It differs from many of them, however, as, in addition to chapters on systematic anatomy, there are chapters dealing with regional anatomy, such as those on the upper and lower limbs, abdomen, thorax, and head and neck. The result of this blending has been to produce a very readable volume in which the subject is presented as a live and not a dead one.

It is very good to find that Professor Frazer is at pains to show: "That text books of Anatomy must be looked on as mere explanatory guides to actual Anatomy, and that they must not be considered to be Anatomy itself." Unfortunately many teachers of anatomy appear to forget this fact, and medical students are still subject to unnecessary tyranny, being required to reproduce at examinations a host of facts that are soon forgotten with the passing of the examination.

Used in the way Professor Frazer recommends, there is no doubt that this manual will become a very popular reference book to medical students, teachers and practitioners.

As might be expected, a good deal of space has been devoted to the embryological aspect of the subject. This adds greatly to the value of the volume and at the same time leads to the making of the only criticism of the work. The embryological descriptions concern three dimensions, while the illustrations of these conditions are only in two dimensions. This fact enormously increases the difficulty of study and converts what should be a relatively easy into a difficult matter. It might be overcome if Professor Frazer could incorporate in a future edition about a dozen stereoscopic pictures. This would be a boon, especially to those students and others who are not so fortunate as to have access to embryological models.

The book incorporates all that is best in previously published anatomical works, and well maintains the high standard set by British anatomists.

[1] "Buchann's Manual of Anatomy, including Embryology", edited by J. E. Frazer, D.Sc., F.R.C.S.; Sixth Edition; 1937. London: Baillière, Tindall and Cox. Double crown 9mo, pp. 1782, with illustrations. Price: 35s.

The Medical Journal of Australia

SATURDAY, JANUARY 22, 1938.

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Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

EFFICIENCY AND THE AUSTRALIAN ARMY MEDICAL CORPS.

AFTER five years' labour, Lieutenant-Colonel T. B. Nicholls, Royal Army Medical Corps, has produced a work entitled "Organization, Strategy and Tactics of the Army Medical Services in War", which is of the highest value to all who are or will be engaged in preparation for war.[1] Such a work has been sorely needed and had to be written by someone. No similar book has ever existed in any country, though a few, especially one hailing from the United States of America, include some of the subjects of the work under review. The best known was the handbook "Field Service Notes for the R.A.M.C.", by Lieutenant-General Sir T. H. J. C. Goodwin, Director-General of Medical Services at the War Office in the latter part of the Great War, and recently Governor of Queensland. This, however, did not cover a fraction of the ground embraced by the present volume; nor could it, for the organization of the medical services reached a pinnacle at the end of the Great War undreamt of in the period when Goodwin's book was published. Nicholls's book is a textbook for the army medical officer, whether administrator or last-joined subaltern. The author has had the advantage of serving in the Great War in all the units of the Royal Army Medical Corps, with one exception; he also gained inside information by being a casualty himself. He has recorded his observations well, for some of his chapters contain valuable information nowhere else available; this applies particularly to the chapter on ambulance trains. But generally the plan has been to collate information scattered in various official manuals and histories and to amplify it.

The book is divided into four parts. First of all the author deals with the general organization and principles of action of an army, the methods of administration of its medical services and some data regarding tasks and resources. He then gives an account of the composition of all the units of the medical service: how they are used and the problems they must overcome, the organization at the base and on the line of communication, with a chapter of invaluable data. When the reader studies the huge and varied organization here portrayed, he is appalled at the task which would have to be faced by someone if war came to Australia. This man would have to create and regulate a similar organization with only a host of amateurs on whom to draw, willing, intelligent and self-sacrificing though they would be. To travel punctually, regularly and safely on a modern railway system makes one wonder that it should ever function so smoothly; yet it would give the traveller pause to picture the years of organization that lie behind this precision, and what would happen if it had to be organized suddenly with but a handful of experts to direct it. A fully functioning medical service in war is comparable, and it must be created in a hurry.

In the third part of his book Nicholls deals with strategy and tactics. There are many people, perhaps a majority, and even some regular soldiers, who

[1] "Organization, Strategy and Tactics of the Army Medical Services in War", by T. B. Nicholls, M.B., Ch.B., with a foreword by Lieutenant-General Sir James A. Hartigan, K.C.B., C.M.G., D.S.O., D.Ch., K.H.P.; 1937. London: Bailliere, Tindall and Cox. Royal 8vo, pp. 386. Price: 10s. 6d. net.

think of a medical service only in terms of pills, dressings and nurses. They do not realize that the medical units of an army are small and often semi-idle, but that at other times they are called on to collect and treat numbers of patients far beyond their capacity either in personnel or equipment, to evacuate them rapidly, and be ready for more. Medical units have to supply efficient and completely up-to-date methods of treatment, to supervise and carry out well considered methods of prevention of disease, and to maintain a supply of medical, hospital and ambulance transport equipment; they have to be in fact, for an army, the hospital, ambulance transport service and drug houses that we know in peace, though hindered by the hostile actions of an enemy and by the circumstances of terrain and climate. To make the best of such a task requires an intimate knowledge of the parts of the machine and of their interaction, of the best arrangement of these parts and of how they may be varied to meet different requirements. This Nicholls tries to explain, and this is perhaps the most valuable part of his book, for lack of proper appreciation of the principles of medical strategy only leads to medical muddles, of which there have been not a few in every war that has ever been fought.

With the last part of the book, that devoted to actual problems and exercises, we are not particularly concerned. What we are concerned with is the immensity of the problem that Australia would have to face in the event of a national military emergency. Members of the community may be patriotic, willing, courageous and even fearless; but if leaders are inefficient the people will be a mere rabble; if leaders have been starved of material for study the people will suffer in like proportion. With the defence policy and armament of Australia this journal should not deal; but it is vitally interested in the army military services. In our opinion, and in the opinion of the Federal Council of the British Medical Association in Australia, the military authorities are not doing their duty to the service concerned. The Federal Council believes, especially when ideas of methods of warfare and their medical involvements are in such a state of flux, that personal acquaintance with individuals, administrative as well as technical, is of much greater value than written instruction from those in high command, and that personal observation is of infinitely greater value than dry-as-dust regulations, even though they be redolent of the army in its more efficient periods. The Council has on more than one occasion recommended to the Federal Government that an officer of superior rank be sent to England to study the latest methods of army medical service, but without result. Advances in every branch of army management have been made since the end of the Great War; but since that time no Australian Army medical officer has been sent abroad for purposes of study. If the Defence Department of the Commonwealth will show that it is prepared to make the leaders of its Army Medical Corps thoroughly conversant with the latest developments in army medical organization, the medical profession of Australia will be satisfied; but not otherwise.

Current Comment.

MORE ABOUT THE DANGERS OF SULPHANILAMIDE.

THAT sulphanilamide is not entirely without toxic manifestations has already been pointed out in these columns. Dizziness, anorexia, nausea, vomiting and cyanosis have all been described by several authors. Even rises in temperature are said to occur, though it must be difficult to separate these from the effects of the infection for which the drug was prescribed. The probability that owing to its benzene ring constituent the drug would sooner or later be found to damage the erythron, has been confirmed by the report of eight or nine instances of hæmolytic anæmia following the use of the drug. S. E. Kohn[1] has recently added another description concerning a child, aged one year, who suffered from an acute streptococcal naso-pharyngitis. The organisms were of the hæmolytic variety. The child was acutely ill; and after a small blood transfusion, given although an initial blood count was almost normal and showed 20,000 leucocytes per cubic millimetre, sulphanilamide therapy was begun. The child received five grains (0·3 gramme) three times a day for twelve doses, a total of one drachm, or 3·6 grammes of the drug. At this point a hæmolytic type of anæmia developed rapidly, and the tempera-

[1] *The Journal of the American Medical Association*, September 25, 1937.

ture, which had fallen when the drug was first administered, began to rise again. The patient's blood was retyped with that of the father, who had been donor for the transfusion, and was again found to be satisfactory. A further crisis followed two days later, and the blood count finally fell to 2,000,000 red cells per cubic millimetre, with a leucocyte count of 47,000 per cubic millimetre, consisting mostly of small and large lymphocytes—a relative agranulocytosis. The blood then rapidly recovered during the next few days, and the child was sent home in fairly good health. To prove the rôle of sulphanilamide in her illness, five grains was repeated three times a day for four doses. The child became restless and irritable, and the temperature began to rise. No further hæmolysis followed.

A new complication is also noted contemporaneously, namely, a toxic optic neuritis after the receipt of the new bacteriocide. Paul C. Bucy, of Chicago, had a patient, a girl, aged sixteen years, who suffered from a chronic osteomyelitis of the upper femoral region, with multiple sinus formation. Bacteriological examination of the discharge from the sinus revealed a mixed infection, including *Streptococcus viridans*. This organism was subsequently recovered by blood culture. The patient had a hectic temperature, and after the failure to improve her condition by repeated blood transfusion, it was decided to give sulphanilamide by mouth. She received a total of 7·5 grammes in two days. Headache and cyanosis were then noted and all medication ceased; but two days later, as she seemed quite better, sulphanilamide was resumed, and a further 7·0 grammes were taken. The same toxic symptoms returned, and no more dye was given for a week, at which time a single tablet was ordered. Next day, the patient complained of dimness of vision. Examination revealed bilateral central scotomata for all colours, and some blurring of the edges of the optic disks. These defects lasted three days and then disappeared, although it took almost a month for the optic disks to resume an entirely normal appearance. It is possible in this case that the ferrous sulphate given simultaneously may have exaggerated the toxic features.

Two further reports accompany the above; these describe skin eruptions occurring during the administration of sulphanilamide. These rashes were more annoying than serious. They took the form of maculo-papular eruptions, with intense itching over areas of the skin exposed to the sun. Large doses of sulphanilamide of the order of 50 grammes of the drug were given in each instance. The appearance of the rash was accompanied by fever, reaching at times to 104° F. There were no accompanying complications of the type known sometimes to occur after sulphanilamide administration, beyond slight cyanosis and hyperpnœa. The rashes lasted for about seven days. In one patient the rash became hæmorrhagic. The explanation of this complication is undecided. Allergy, interaction between a heliosensitivity created by the drug or its derivatives and hæmoglobin are suggested.

There is no question but that the above described complications are very rare following "Prontosil" or allied therapy, as the drug is being consumed in enormous quantities the world over. With the exception of the cutaneous lesions, which are not dangerous and which seem to be related to heavy dosage of the dye, the remaining mal-effects seem to occur in persons sensitized by an inherent idiosyncrasy.

The warning to omit other drugs capable of causing methæmoglobinæmia and the sulphates, and to give test doses, controlled by serial blood counts, is good advice from a hospital point of view. For the general practitioner the position is more difficult; he may be satisfied with the great rarity of untoward sequelæ, but in spite of his inadequate facilities he should not omit the precautions that would be adopted if his patient was in hospital.

MITRAL STENOSIS DUE TO TRAUMA.

In an interesting report from the Derbyshire Royal Infirmary H. Barber points out that valvular disease due to trauma must be divided into classes: (*a*) lesions which are the result of strain, and (*b*) lesions which are the result of a direct blow on the chest wall. He states that it has been proved experimentally after death that a blow may rupture a valve. He describes a case in which mitral stenosis resulted from trauma to the chest wall.[1] The diagnosis was confirmed by G. R. Osborn, who made the *post mortem* examination. The primary lesion of the mitral valve was caused when the patient, a Class A soldier, aged thirty-two years, was blown up and buried by a shell explosion while on active service. When he regained consciousness after a few days he found that he was very short of breath and had an irregular heaving action of the heart. He had no wounds or external bruises. After several years signs of mitral stenosis became obvious and the patient had to lead a very restricted life. He died of pneumonia twenty-two years after the trauma occurred. From *post mortem* appearances it was concluded that the stenosis followed the healing of a relatively large hæmatoma.

ALFRED WALTER CAMPBELL.

In this issue are published several appreciations of the late Alfred Walter Campbell. The readers of this journal are under a lasting debt to him—for upwards of eighteen years he abstracted current literature on neurology and psychiatry and was more than generous in reviewing books and in giving advice and help whenever he was approached. He was one of those who came to the assistance of the late Henry William Armit in the difficult days of 1914 when this journal was being established. This acknowledgement is made in appreciation and gratitude.

[1] *Guy's Hospital Reports*, October, 1937.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Laboratory Findings in Amœbic Dysentery.

R. H. KAMPMEIER AND E. HAROLD HINMAN (*The Journal of Laboratory and Clinical Medicine*, July, 1937) review the results of laboratory tests in a series of 400 cases of amœbic dysentery occurring in the State Charity Hospital, New Orleans. The parasite was found in each case, and ulcers were seen in 87% of patients examined by the proctoscope. It appeared from their experience that in clinically active cases the diagnosis might be readily established in a high proportion of cases either by a single examination of the stool or by proctoscopic examination. The authors quote the results of Brown, who, in a series of over 500 cases, was able to demonstrate the organism in 68·2% by a single examination of the fæces, and in 98% by examination on three successive days. A secondary anæmia was found to be present in the majority of cases. In only about one-quarter of those examined was the leucocyte count above 10,000 cells per cubic millimetre, and differential cell counts showed no characteristic findings. Gastric analysis was performed in 40 cases; in 25 of these either achlorhydria or hypochlorhydria was found. No definite conclusions could be drawn from radiological study of the intestinal tract.

Cycle of Avian Malarial Parasites in Endothelial Cells.

S. P. JAMES AND P. TATE (*Transactions of the Royal Society of Tropical Medicine and Hygiene*, June 25, 1937) illustrate the recently discovered cycle of avian malarial parasites in reticulo-endothelial cells. *Plasmodium gallinaceum*, like other members of the family Plasmoliidæ, has a schizogonic cycle of development in the erythrocytes of the vertebrate host, and a sporogonic cycle in the insect host (*Stegomyia fasciata*). In addition, it has a hitherto unrecognized schizogonic cycle in monocytes and reticulo-endothelial cells of the spleen, liver and kidneys, and particularly, in certain cases, in the endothelial cells lining the capillaries of the brain. The occurrence, in human parasites, of such a cycle in tissue cells instead of in red blood cells, has been previously postulated to explain why quinine was ineffective in preventing malarial attacks and relapses and recurrences of the disease. The results of prophylactic and clinical trials with quinine and synthetic preparations on chickens infected with *Plasmodium gallinaceum* seemed to show that the endothelial cell cycle of the parasite might help to explain some of the unsolved problems of malaria. Chickens temporarily cured of the peripheral blood infection by quinine had later died as the result of the development, in the endothelial cells of the cerebral vessels, of large schizonts, which completely blocked the capillaries. It does not seem correct any longer to define the Plasmoliidæ as a family in which the whole of the vertebrate cycle of development occurs in the circulating red blood cells.

Apical Lung Scars.

J. DAWSON AND W. SUSMAN (*The Journal of Pathology and Bacteriology*, November, 1937) write about apical lung scars and their relationship to siliceous dust accumulation in non-silicotic lungs. The series consisted of 94 cases out of an original group of 107, from which 13 cases of subacute or chronic pulmonary disease had been excluded. The original 107 cases formed as far as possible a consecutive series. Occupation and disease incidence were varied and none showed any special preponderance. The age incidence corresponded roughly with that seen in any general series of *post mortem* cases. The proportion of males to females was as five to four. In the 94 cases the lungs were free from gross pulmonary disease; they were examined histologically and by microincineration. Siliceous matter was present in association with carbon in all cases. In general the amount of siliceous dust increased with age and was least in country dwellers. Most dust was found in the apical regions and least in the lower lobes. There were 40 cases with apical scars without evidence of healed tuberculosis (Type A scars) and six cases with apical scars in which evidence of healed tuberculous was found (Type B scars). A definite relationship existed between siliceous dust accumulation in the upper part of the lung and the presence of Type A scars. Silicotic nodules, in varying stages of development, were present in 13 cases. These belonged to the later age groups and were cases with a high silica content in the lungs generally. Since evidence of past tuberculosis is lacking in Type A scars, and since silica is known to produce fibrosis, the authors conclude that the development of these scars is secondary to the accumulation of siliceous matter at the apex.

The Red Cells in Acholuric Jaundice.

JANET M. VAUGHAN (*The Journal of Pathology and Bacteriology*, November, 1937) has studied the character of the red blood cells in a series of cases of acholuric jaundice both before and after splenectomy. She found increased red cell fragility in sodium chloride solutions in 35 cases of acholuric jaundice and increased spherocytosis in 25 of 27 cases examined. She holds that megalospherocytosis as well as microspherocytosis may occur in acholuric jaundice. Following splenectomy, increased fragility invariably persisted; but spherocytosis was lost in 50% of patients. The author suggests therefore that both erythropoiesis and splenic function are at fault in this disease. She found a high degree of correlation between mean corpuscular thickness and median corpuscular fragility, both in acholuric jaundice and in other conditions before splenectomy. Since fragility remained abnormal after splenectomy while spherocytosis returned to normal, the author concludes that increased spherocytosis is not the fundamental abnormality present in the acholuric red cell.

The Portal of Entry of the Poliomyelitis Virus.

J. A. TOOMEY (*The Journal of the American Medical Association*, August 7, 1937) renews his opposition to the conception that the virus of infantile paralysis commonly enters the body by way of the olfactory nerves. He alludes to the difficulty of producing the disease by intranasal instillation of the virus in monkeys, and to the rarity of contagion amongst monkeys and in hospitals. He has achieved what has been held to be impossible in producing poliomyelitis in macaques by the intravenous injection of virus and by its introduction into the alimentary canal after section of the olfactory tracts and after cauterization of the olfactory epithelium with zinc sulphate. He revives his theory that the portal of entry of the virus is the diseased gastro-intestinal tract, whence it passes along sympathetic and parasympathetic nerves to the central nervous system. He states that lack of vitamin *D* (which is one cause of disorganization of nerve myelin) causes monkeys to develop poliomyelitis very readily when the virus has been "placed in the intestine".

J. F. LANDON (*Ibidem*), discussing Toomey's theory, points out that very little microscopic change is found in the olfactory bulbs of human subjects dead of poliomyelitis.

The Pathogenesis of High Blood Pressure.

HARRY GOLDBLATT, who in 1934 produced persistent arterial hypertension in dogs and monkeys by narrowing the renal arteries with silver clamps, reports experiments showing that this hypertension is due to a humoral mechanism and not to a nervous mechanism initiated by renal ischæmia (*Annals of Internal Medicine*, July, 1937). The arterial constriction does not cause hypertension when accompanied by occlusion of the renal veins, presumably because a pressor substance is excluded from the circulation. When one renal artery has been constricted the elevated blood pressure falls to normal when the ischæmic kidney is excised. Further experiments show that some suprarenal cortex must be present or cortical extract must be administered

before constriction of the renal arteries will produce high blood pressure.

Epithelial Metaplasia.

K. McCULLOUGH AND G. DALLDORF (*Archives of Pathology*, October, 1937) in experiments on rats have studied the effects of theelin, mechanical irritation and vitamin *A* deficiency on epithelial metaplasia. Vitamin *A* deficiency is the primary essential condition for metaplasia; theelin and mechanical irritation act solely as secondary factors.

MORPHOLOGY.

Foramen of Magendie.

J. T. WILSON (*Journal of Anatomy*, July, 1937) describes the nature and mode of origin of the foramen of Magendie. Such a structure he proves to be normally present in the human adult as an aperture of varying size in the lower, or caudal, triangular portion of the roof of the ventricle. The results obtained from an examination of a series of transverse sections across the *medulla oblongata* and fourth ventricle of a fœtus 129 millimetres in length, that is, of the fifth month, coincided with the earlier observations of Blake, who was the first to recognize and elucidate fully the real nature of the foramen of Magendie as the aperture of an actual saccular evagination of the ventricular cavity. Atrophic degeneration occurs in this saccular diverticulum and considerable areas of the wall of the cavity disappear. The cavity then becomes incorporated with the large subarachnoid cistern in this situation, but the neck of the saccular evagination is preserved more or less completely as the adult foramen of Magendie with its reflexed margins.

Major Subdivisions of the Marsupialia.

A. A. ABBIE (*Journal of Anatomy*, July, 1937) discusses the major subdivisions of the Marsupialia, with especial reference to the position of the Peramelidæ and Cænolestidæ. The presence of a *fasciculus aberrans* in the anterior commissure of the brain in Diprotodontia, and its absence in the Polyprotodontia, provide an exact distinction between the two great groups of marsupials. In the vast majority of cases the state of the dentition constitutes an accurate guide to the condition of the anterior commissure. The teeth, therefore, supply the most valuable external feature upon which to base any classification of the Marsupialia. The condition of the *pes* does not present such a fundamental criterion. The condition of the anterior commissure is the surest guide to classification if doubt arises in the examination of recent material. The author concludes that those living marsupials of equivocal status as regards dental and pedal characters, the Cænolestidæ and Peramelidæ, must, in view of their lack of a *fasciculus aberrans*, be assigned to the Polyprotodontia.

Lymphatics of the Stomach.

J. H. GRAY (*Journal of Anatomy*, July, 1937) describes a method of filling the lymphatics of the stomach of the human fœtus by injecting the arteries with a barium suspension. He finds that the subserous plexus of stomach lymphatics is a rich plexus with an extensive anastomosis between the vessels running towards the two curvatures. The continuity of the subserous plexus maintained by Jamieson and Dobson is proved. This finding was not that held by the French investigators Cunéo and Delamare, who stated that there was a very poor anastomosis in the subserous plexus between the vessels draining towards the chief groups of gastric lymph glands. Another point of interest stressed by the author concerns what occurs at those points where the direction of lymphatic flow is reversed either towards the greater curvature or towards the lesser curvature. His specimens show that any one lymphatic vessel which runs across both of the two main drainage areas contains two valves at a greater or lesser distance apart and facing in opposite directions. This finding is explained in terms of Sabin's theory of the development of lymphatics by sprouting.

Pars Intermedia of the Hypophysis.

P. J. GAILLARD (*Acta Neerlandica Morphologiæ*) describes an experimental investigation into the origin of the *pars intermedia* of the hypophysis. Explants from the anterior lobe of the hypophysis of three months old rabbits were cultivated with explants from the posterior lobe of the same animals in one culture drop. In this drop the two cultures were placed side by side. In that part of the anterior lobe explant which was lying close to the tissue of the posterior lobe explant, an intermediate-lobe-like structure was found, the chromophil cells disappearing and cysts being formed there. This special differentiation occurred in 85% of the experimental cultures. It was not observed if either were cultivated with explants from the testis, thyreoid gland, suprarenal gland, placenta or *corpus luteum*, or with osteogenetic cells from the primordium of the frontal bone of 16 days old chicken embryo. From a study of the literature and on the ground of the culture experiment, the author suggests that the conception of Cowdry, who considered the structure of the intermediate lobe to be at least partially due to vessel deficiency, can hardly be maintained. The possibility is suggested that the structures of the intermediate lobe and of the *pars tuberalis*, as they occur in man and in various animals, are due to a direct influence of the *pars nervosa* and the infundibular body, respectively, on that portion of the *pars oralis* which is lying in contact with them.

Uterine Growth Resulting from Chronic Distension.

S. R. M. REYNOLDS AND S. KAMINESTER (*The Anatomical Record*, October 25, 1937) have studied the rate of uterine growth resulting from chronic distension. They used rabbits from which the ovaries had been removed and have conducted experiments of two types. In the first, paraffin pellets have been introduced *per vaginam* into the uteri; in the second type of experiment fluid was allowed to accumulate in the lumen of the uterus between two ligatures placed about an inch apart. The authors describe the growth changes that take place. After insertion of paraffin pellets, growth changes, including both hypertrophy and hyperplasia, are completed in less than four days. When distension is caused by progressive accumulation of fluid within the uterus, the growth response is proportional to the degree of distension. In the latter type it was found in some instances that the percentage increase in the size of the uterus approached that which takes place during normal pregnancy in the rabbit.

Anterior Hypophysis of the Guinea-Pig.

HADLEY KIRKMAN (*American Journal of Anatomy*, July, 1937) records a cytological study of the anterior hypophysis of the guinea-pig and a statistical analysis of its cell types. The secretory cycle of guinea-pig anterior pituitary chromophiles is characterized by progressive cytoplasmic and nuclear changes which are microscopically demonstrable. Although basophiles and acidophiles appear at about the same time in the anterior hypophysis of the fœtal guinea-pig, the former are more precocious in their development. There is a decrease in the relative number of basophiles with some increase in colloid during œstrus. At this time there is an increase in the relative weight of the pituitary but no change in its absolute weight. The author states that the production of colloid by basophiles of the *pars intermedia*, as well as by cells of the *pars anterior*, during late pregnancy and soon after parturition, is incompatible with the concept that the acidophile is the only secretory cell type in the pituitary gland and is solely responsible for the production of colloid. No transformation of *pars intermedia* cells into acidophiles occurs. After gonadectomy, there occurs an increase in the relative number and size of basophiles in the anterior hypophysis of the guinea-pig, as in the rat. A decrease in the size and number of acidophiles also occurs. Some increase in colloid occurs. Gonadectomy changes are more marked in the female than in the male.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on July 10, 1937, PROFESSOR R. MARSHALL ALLAN, the President, in the chair.

Surgery in the Common Bile Duct.

DR. K. ROSS read a paper entitled "Personal Experience in the Surgery of the Common Bile Duct" (see page 160).

MR. BALCOMBE QUICK opened the discussion by stating that it had been a very real privilege to listen to Dr. Ross's paper. The first slide shown was of great value in that it made plain that in a considerable percentage of cases stones in the common duct were attended neither by jaundice nor by colic, and that the text-book picture of Charcot's intermittent fever was by no means commonly met. Dr. Ross had very wisely limited his paper to one aspect of gall-bladder surgery—the difficulties associated with the common duct. This might be looked upon as the knife-edge of the balance in which success or failure in the surgery of the gall-bladder often hung. There was no room for argument as to the necessity in the vast majority of cases for finding and removing the common duct stone. The unpremeditated leaving of a calculus there exposed the surgeon to obloquy and placed the patient's life in jeopardy.

The difficulties met with in this region were either man-made, as, for instance, injuries to the common duct at operation, or were sent by Providence to try the surgeon. Those of the latter type were the less appalling, but they were nevertheless sufficiently real. Regarding congenital abnormalities of the ducts, Mr. Quick had been impressed with the discrepancy between the alleged frequency with which they were found at autopsy and in the dissecting room and his findings on the operating table. With the exception of certain trivial errors, such as double cystic arteries or a cystic artery which was a little in front of or a little behind the usual position, or minor peculiarities of the cystic duct, as, for instance, an unusually low junction with the common duct, he had met with no undoubted examples. It had occurred to him that the question of congenital abnormality was invoked at times to gloss over the fact that the surgeon had failed to see something that he might have observed earlier.

Mr. Quick then went on to consider when and how the common duct should be explored for a possible stone. He said that the indications for exploration as laid down by Maingot were to be highly commended. When the gall-bladder harboured a single non-facetted stone of the cholesterin type the surgeon could pass by the common duct with an easy mind, even if it appeared rather large. When any doubt existed, however, the duct should be explored, for such exploration added nothing at all to the gravity of the operation when the patient was not particularly ill. Mr. Quick said that the instruments he used for the purpose were suitably sized Hegar's dilators, Desjardin's forceps, and sometimes the old Watson-Cheyne dissector. All flexible probes were as reliable as broken reeds. It could be very difficult to decide when the complete patency of the common duct through to the duodenum had been proved. The presence of a stone in the lower part of the common duct or at the ampulla of Vater could not be determined with certainty by palpation through the duodenal wall or from the pancreatic aspect. Even when the exploring instrument appeared to pass freely into the duodenum, the finding of the slightest trace of *débris* or pigment adhering to it or following its withdrawal was of the greatest significance and demanded that the question should be settled beyond cavil. In spite of everything it might be difficult or impossible to prove a fairway to the duodenum by duct exploration from above. In these circumstances Mr. Quick thought the surgeon more diffident than he should be about opening the duodenum. Trans-duodenal choledochotomy was not the difficult or dangerous operation some might think. An instrument passed down the common duct through a supraduodenal incision would impinge either upon a stone or upon the ampulla, and an incision through the duodenal wall at this level would enable any doubts to be settled. He preferred a transverse incision of the duodenum to the usual longitudinal one, as it lay in the direction of the vessels and consequently there was much less bleeding. He had adopted this procedure on three or four occasions, and he had had no reason to regret it. He recalled a patient upon whom he had performed a cholecystectomy and exploration of the common duct and whom he had again fruitlessly explored for recurrent attacks of pain. He had explored the patient yet again by opening the common duct in its supra-duodenal portion by passing a sound until it was arrested at the ampulla and then by incising the duodenum at this level. Four or five small stones were then revealed in what was virtually a diverticulum lying in a dilated pancreatic duct; but whatever they were, it would have been quite impossible to deal adequately with this situation by any other method.

Mr. Quick also referred to one small procedure which he had found of considerable help and comfort to him in difficult cases; it was not really relevant to the common duct problem, but to cholecystectomy after an exploration of the common duct. Through the opening into the duct above the duodenum an instrument—a rubber catheter or a sound of some sort—was passed up into the hepatic duct towards the liver. In the subsequent dissection to free the cystic duct and in the application of forceps to it and to the cystic artery the presence of an instrument in the ducts as described might prove of the greatest value. The absence of such a guide had on one occasion provided a distressing experience: A large pair of forceps had been closed completely across the common duct and had been left thus for quite ten minutes before the position was recognized. When the forceps were removed the duct walls were teased apart and made to open up well. The patient was young; the operation was performed some twelve years previously, and she had not become jaundiced. Mr. Quick said that this experience exemplified the powers of rehabilitation that resided in the common duct.

Mr. Quick then passed on to consider the management of those unfortunate patients in whom portion of the common duct had been removed, although his experience of such cases had been fortunately limited. If the damage was recognized and the repair was carried out at once over a Macarthur tube the prognosis was not so bad. Even in long-standing cases the technique appeared to be successful in a number of instances. It had seemed to him, however, as a result of one experience, that too much reliance should not be placed upon the powers of regeneration of the epithelial lining of the duct. The gap to be bridged and the consequent degree of tension were the determining factors. When both were minimal the Macarthur technique was likely to be satisfactory. These conditions were absent in the patient in question, in whom from 1·9 to 2·5 centimetres (three-quarters to one inch) of the duct appeared to have been removed. Repair over a Macarthur tube was carried out, but with an undesirable degree of tension. The tube had remained *in situ* for eighteen months before being passed. The patient then remained fairly well for two or three years before stenosis of the duct caused fatal jaundice and cholæmia. In such a case, when any question of undue tension was involved, Walton's duodeno-hepaticostomy was to be preferred; and it should have been carried out in the instance Mr. Quick had quoted.

DR. W. A. HAILES said that the subject under discussion was one in which he had been very interested in association with Dr. Cowen at the Royal Melbourne Hospital. He touched first on the question of anomalies in the common duct which had been raised by Mr. Quick. He had never met with one, though a well-known specimen of an absent cystic duct appeared to be explainable by suppuration, destruction of tissue and a double fistulous communication with the hepatic duct above and the common duct below. Dr. Hailes said that some surgeons opened the common duct in every case, but he could not subscribe to that procedure; each case should be dealt with on its

merits. Dilatation of the duct, the presence of pus in it or any abnormal appearance of the bile would be indications for opening the duct. He admitted that he had been unable even to approach some of the very low mortality figures in cases in which the common duct was opened for stones. To reduce the mortality, pre-operative preparation required careful attention, and as little as possible should be done at operation. He often left the gall-bladder and was content to drain it. In spite of attention to these points he could not approach some of the published statistics.

Dr. Hailes then said that he had come to the conclusion that if the presence of a stone in the common bile duct was suspected the patient should not be kept too long before operation. Pre-operative preparation to combat acidosis was usually desirable. He remembered a patient who had had gall-stone colic over a period of years and who had had an attack of biliary colic suddenly and in less than thirty-six hours was in a state of acidosis. The patient was given glucose and it appeared in the urine; eighty units of insulin daily were given with the glucose before acidosis was controlled. At operation a stone was removed from the common duct, and the patient had left hospital sugar-free and on ordinary diet. It was necessary to make quite sure of the pancreatic metabolism, and if the glucose metabolism was correct then the operation should be performed. While it was preferable to operate in the absence of jaundice, the tendency to delay until jaundice had disappeared could be overdone. He referred to a patient who was jaundiced with the first attack of biliary colic. The patient was prepared by being given sugar, and the sugar metabolism was correct, but three days from the onset of jaundice the temperature rose to 40·5° C. (104° F.), and death from acute cholangitis took place in two or three days. After the rigor and the elevation of temperature the condition did not justify any interference. He had been told that it was fortunate that he had not operated on the patient, but he did not know whether this was so. He thought that surgeons had been waiting too long before operating when a stone in the common duct was suspected. The risk of stenosis from the opening of a big common bile duct was negligible, Dr. Hailes said, but if the duct was small and was drained there was a risk. He had had a patient upon whom he had operated for gall-bladder stones. The tissues were very œdematous and he had performed cholecystectomy. The patient's condition was satisfactory for three weeks, and then very severe pain necessitated the administration of morphine. A month after the previous operation he had opened the common duct, but had not found a stone; drainage immediately and completely relieved the pain. He had stitched in a small catheter, and when the tube came out drainage continued for a few days; but the patient was discharged from hospital one month from the second operation and then seemed to have quite recovered. Three years later a stricture of the common bile duct had developed. Dr. Hailes thought, therefore, that it was a mistake to stitch a tube into a small common bile duct; drainage down to it was all that was necessary. He had had experiences of operating for strictures twice. On one occasion he had used the Walton method and the patient had died. The technical difficulties were great and a duodenal fistula had developed. On the other occasion he had followed the Mayo method and no fistula had formed. He thought that the Mayo method was much to be preferred, and he intended to use it in future. Dr. Hailes, in conclusion, emphasized the point that the pancreatic metabolism had to be considered and that operation should not be performed if it was unsatisfactory, but should not be delayed if it was in order.

DR. S. O. COWEN said that he rose to contribute to a surgical discussion with a physician's temerity, but Dr. Hailes had provoked him to speak. They had had a series of cases together and his interest in the subject had been aroused. In a large general hospital a considerable share in the diagnosis and in the preparation of the patient for operation fell to the lot of the physician. Whatever faults and failures they had, surgeons were extremely honest. He had admired Dr. Ross's paper for its fearless honesty, and conditions had not been ascribed to causes he had not proved. Dr. Cowen asked how often obstruction of the common duct occurred without either jaundice or biliary colic. He had had a number of such cases which had resembled catarrhal jaundice, but he thought the condition was very rare without either pain or jaundice. In the winter and spring of 1935 there had been a considerable number of patients with infective hepatitis which was quite common in children and young adults and showed very distinct epidemic tendencies. Dr. Cowen referred to the diagnosis of stone in the common duct and other biliary conditions. The notion was prevalent that it was dishonest of the surgeon to operate on the biliary tract if cholecystographic examination revealed no abnormality. Dr. Ross had mentioned the achalasia of the sphincter of Oddi that had been found at operation. There was a certain number of patients with classical biliary colic and normal cholecystograms. Dr. Cowen believed that these should be operated on. It should be frankly and freely admitted that in the presence of anatomico-pathological lesions of the biliary tract medical treatment was absolutely useless.

Dr. Cowen thought that the question of preparation of these patients for operation was an interesting one. He agreed with Dr. Hailes that operation was often too long delayed. A cholæmic patient should be quickly put into a reasonable state for operation by means of copious amounts of fluids together with glucose and insulin. There was no need to worry about glycosuria so long as ketosis was absent. He believed that the administration of calcium had something to do with lessening the tendency to hæmorrhage. In these obstructive cases, however, the serum calcium was usually quite up to normal, and yet calcium chloride lessened the tendency to bleed. The serum calcium might be in such a chemico-physical combination that it was not all available for clotting. He would like to know whether the surgeons were very keen on calcium therapy for these patients. One eminent authority had said that abundant fluids were as good as calcium.

Dr. Cowen stated that it fell to the lot of the physician to see a certain number of the surgeon's failures. To remove the gall-bladder in biliary obstruction without ascertaining the cause of the obstruction seemed to him to be a dreadful thing to do. The resulting problems of treatment were very difficult. It should be axiomatic that the gall-bladder should not be removed without the cause of obstruction being ascertained. All physicians had been impressed by the good prognosis in carcinoma of the head of the pancreas with an efficient anastomosis between the gall-bladder and the stomach, yet chronic pancreatitis was invoked as a cause of obstruction of the common duct.

DR. VICTOR HURLEY appreciated Dr. Ross's paper as being an actual record of work done which was a valuable basis for any discussion. With regard to the diagnosis, he thought that the frequency of colic and pain in the presence of common duct stones had been greatly overstated in the text-books. He had not estimated his figures, but he thought that the number of patients without colic or pain would perhaps be 20% to 25%. It should be very widely known that common duct obstruction could occur without either. The way in which the history was taken was of importance, and experience counted for much in this regard. The patient should be closely cross-examined, as in this way a very suggestive history could be obtained in otherwise doubtful cases. Dr. Hurley preferred to pin his faith to a strongly suggestive history rather than to a doubtful cholecystogram. If the history was right, even despite a normal cholecystographic examination, the surgeon was perfectly justified in operating. Graham had said that cholecystographic examination should be repeated in a few days if the first gave no results. In cholesterosis of the gall-bladder it was sometimes necessary to open the gall-bladder to verify the diagnosis, as otherwise the condition might escape recognition. In patients with jaundice the difficulty was that radiologists were unwilling to make a cholecystographic examination while the jaundice was present.

Discussing the question of when to operate, Dr. Hurley said that he was operating earlier and earlier on patients

with jaundice. If he could forecast in which cases the jaundice would subside, the surgeon would be in a better position in deciding when to operate. It was admittedly safer to operate when the jaundice had subsided, but some patients passed from one attack into another and might have several in a few days. In such cases the signs of cholæmia and toxæmia became more pronounced and the patient's condition deteriorated. Broadly speaking, the results were likely to be better if operation was performed earlier.

Dr. Hurley then discussed the circumstances in which the common duct should be explored. He was more conservative than the other speakers on this matter. If there was clear evidence in the history and if signs of obstruction were found at operation he explored the duct. If the duct was not dilated, if careful palpation revealed nothing, and if the history did not indicate the procedure, he did not open the common bile duct. He had found it easier and more reliable to palpate the common bile duct and the head of the pancreas from the other side of the table. He got his assistant to feel and he himself felt the area, and if they were both satisfied he regarded that as sufficient evidence. He thought that opening the common duct unnecessarily did add slightly to the risk of the operation. A small vessel running across the duct was frequently cut when an incision was made into it, and it was amazing how troublesome this could be in a stout patient. Then again, if a very small stone was missed the patient might pass it. On at least three occasions he had seen a stone that had been recovered in the stools in such circumstances.

Dr. Hurley said that these patients were frequently very ill and there were no reliable tests of liver function, nor was there an easy escape as in urinary operations. In the latter, the patient's renal could readily be estimated, and minor procedures, such as the tying-in of a catheter or suprapubic drainage, could be used to relieve him. There was no short cut, however, in biliary surgery. He agreed with Dr. Hailes that a routine cholecystectomy should not be performed, or should the operation be extended unnecessarily in these sick patients, but if the surgeon was in any doubt about the patient's condition he should be content to remove the stones and to drain the gall-bladder.

Dr. Hurley then considered the circumstances in which the common duct should be drained. He found himself dispensing more and more with drainage and suturing the incision in the duct. The exploring instruments that he used after any stones had been removed were Hegar's dilators, the ordinary curved common duct forceps and Clutton's urethral sounds. It was difficult to get straight Hegar's dilators to make the curve as well as a urethral sound. He had the feeling that a patient whose bile was conserved and allowed to pass into the duodenum did very much better than the one in whom the bile was allowed to drain externally. If a number 8 or 10 metal sound could be passed the common duct could be safely closed without drainage. He did not think that collecting the bile drained externally and putting it back into the rectum did any real good.

Dr. Hurley said that the diagnosis between chronic pancreatitis and malignant disease of the head of the pancreas had to be made on what could be felt at operation, and the diagnosis could not always be made correctly. He referred to a patient who had died as the result of hæmorrhage from varicose ulcer after surviving for six years after the diagnosis at operation of malignant disease of the head of the pancreas.

With reference to repair of the common duct, Dr. Hurley stated that he had encountered two cases. In one the common duct had been divided at an operation at which everything was apparently going well. Two openings were seen in the forceps instead of one. For three or four days after operation the patient had had deepening jaundice, and at a subsequent operation on the fifth day the damage was found at the junction of the right and left hepatic ducts with the common bile duct. A successful restoration by suture was carried out and the patient had remained well for over a year, when evidence of stenosis of the common duct appeared, and the patient died as if from cirrhosis of the liver three or four years later. In the second experience he had found the lesion at the same place and he had not been satisfied with the repair as a satisfactory junction was not possible, but nevertheless the patient had done well. Two and a half years had elapsed since the repair and the patient was now quite well. This experience had increased his respect for the capacity of the duct to be rehabilitated. In conclusion, Dr. Hurley mentioned a minor point in technique. He said that these patients were often very stout and the incisions were very difficult to close. The sutures tended to pull through the posterior sheath of the rectus muscle owing to the horizontal direction of their fibres. He, therefore, made the incision in the posterior sheath of the rectus muscle from the top left-hand corner to the bottom right-hand corner. When suturing he made mattress stitches on the side nearer him. By these means he found that the closure of the abdomen was carried out more easily and without tearing.

DR. JOHN KENNEDY said that some ten years earlier he had operated upon a patient for stone in the common duct, and on finding a mass present in the pancreas he made an anastomosis. The patient had had a "++++" reaction to the Wassermann test. On another occasion he had found a gumma. Speaking of when to operate, he agreed with Dr. Hurley and Mr. Quick. The surgeon should not rush operation, but should treat each case on its merits; if the patient was not improving, operation should be performed as quickly as possible. Dr. Kennedy palpated with the left hand and could feel the head of the pancreas, but he was not sure that he could feel a stone. He agreed that closure was difficult, but he thought that a mistake was made in pulling the rectus muscle outwards. He had seen a patient recently with a perfect scar upon whom the late Hamilton Russell had made a Mayo Robson incision when he operated twenty years previously.

PROFESSOR R. MARSHALL ALLAN, on behalf of the Victorian Branch, congratulated Dr. Ross and invited him to close the discussion.

Dr. Ross, in reply, said that he did not think that there was very much he was called upon to answer. He was afraid he had been misunderstood in the matter of catarrhal jaundice; what he had meant was that when the diagnosis of catarrhal jaundice was made, the surgeon should make a mental reservation and be prepared to review the diagnosis later.

A MEETING of the Victorian Branch of the British Medical Association was held at Warragul on October 2, 1937, DR. J. P. MAJOR, Senior Vice-President, in the chair. Part of the meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the West Gippsland Hospital.

Rheumatic Heart Disease Associated with Pulmonary Tuberculosis.

DR. J. M. ANDREW showed a girl, aged sixteen years, whom he had first seen in February, 1937. She had been admitted to a metropolitan hospital in September, 1936, with rheumatic pancarditis and had been an invalid until December, 1936, and even when he saw her in February, 1937, it was obvious that she had gross heart failure. The apex beat was in the sixth interspace and towards the axilla, and her ankles were œdematous. On examination of the chest he found evidence of congestion of the right lung and of the upper part of the left lung, and crepitations and râles were audible. These abnormal findings had persisted despite improvement of the heart condition in hospital. The skiagram had confirmed the gross enlargement of the heart, but after two weeks' rest in bed there had been definite improvement. Several sputum examinations and antral investigations were carried out, no abnormality being detected, and after two months in hospital the patient went home. Two weeks later she had had an intense hæmoptysis and shortly afterwards two

more with fairly continuous sputum, though no tubercle bacilli could be found in it. Further skiagrams of the chest were made and the chest was examined by means of the fluoroscopic screen, but at that time Dr. Andrew had been unable to be sure that the changes seen were tuberculous. Tubercle bacilli had been found in the sputum, however, three weeks before the meeting and again subsequently. While the patient was at complete rest she was not feverish, but she became pyrexial on slight exertion. Dr. Andrew said that he had shown the patient to get expressions of opinion concerning the prognosis and the treatment of the lung condition.

DR. M. D. SILBERBERG said that the prognosis had to be considered from three aspects: (*a*) that of the heart condition alone, (*b*) that of the lung condition alone, and (*c*) that of the heart and lung conditions and the influence of one on the other. He feared that the serious heart lesions would eventually prove fatal and that the prognosis was largely a matter of expression of opinion concerning the duration. With reference to treatment, very prolonged rest was indicated over a period of at least twelve months. So far as specific treatment was concerned, nothing was likely to do the heart much good except rest; the rheumatic process might be influenced by the intermittent exhibition of salicylate of sodium and by courses of injections of streptococcal vaccine in minute quantities; the effect of sulphanilamide drugs might prove of interest. If they had not had Dr. Andrew's assurance of the presence of tubercle bacilli in the sputum, Dr. Silberberg said that he would have regarded the condition as mitral disease with lung changes simulating those of tuberculosis. While the prognosis was serious, it should not be forgotten that substantial improvement might occur.

DR. C. H. FITTS said that though it might sound as if he was being wise after the event, he would like to make some observations on the appearances in the films. In the early one it could be seen that the heart was enlarged, presumably by acute dilatation, and it was likely that the lung was acutely congested. If the congestion was the only lesion, the evidence would remain limited to a butterfly area around the hila, but in this case the areas of diminished transradiancy extended into the upper part of the lungs; and this was more obviously so in the right lung than in the left one. Dr. Fitts agreed that prolonged rest was the rational treatment; he did not consider the patient suitable for any operative procedure on the lungs; nor was she a suitable patient for sanatorium treatment, though she should be in a hospital such as the Austin Hospital in a tuberculosis ward if the home conditions were not satisfactory.

Alcoholic Gastritis and Cirrhosis of the Liver.

DR. ANDREW also showed an elderly man who in his early life had been a miner in Tasmania. At the age of eighteen years he had had an attack of typhoid fever, which was followed by backache and pain over the lower part of the spine. In 1929, at the age of forty-eight years, he had come under notice on account of persistent cough; and though examination of the sputum revealed nothing, the skiagram indicated the presence of pulmonary fibrosis. At that time it was known that the patient had chronic gastritis from excessive indulgence in alcohol. Three years later he had fallen from a height of eighteen feet on to his right side and had injured his shoulder and back; bruising appeared on the right shoulder, forearm and arm. On July 19, 1937, after a motor-car accident, he had been exposed to the weather for some time in a drunken state, and was admitted to hospital on August 8. He was very ill with persistent vomiting, cough and sputum, and intense radiating pain in the back. By August 10 the temperature had reached 39·5° C. (103°F.); he had a filthy tongue and offensive breath and was in a uræmic condition. No tubercle bacilli were found in the sputum; he vomited for ten days and had an irregular pyrexial temperature. The blood urea was estimated at 61 milligrammes per 100 cubic centimetres of blood, but the urea concentration was over 4%. Examination of the gastric contents revealed achlorhydria with complete absence of free hydrochloric acid, though the total chlorides were normal; no lactic acid was found, but a great deal of mucus was present. In a skiagram of the chest the hilous markings were excessive, with radiating fibrosis; in a film of the spine a lesion could be seen affecting the tenth and eleventh thoracic bodies, with destruction and collapse chiefly on the right side. The dental condition was one of gross sepsis; he had had a number of extractions in 1930, but the lower teeth especially were in very bad condition. The pain in the back radiated to the right side, and he required sedative drugs for its relief; a jacket was made to support him, which he refused to wear; but it was ascertained that splinting relieved the pain. He was still running a mild but irregular febrile course when he left the hospital one week before the meeting, and when he was seen by Dr. Andrew on the day before the meeting it was apparent that he had been indulging in his favourite beverages. Dr. Andrew added that he thought there was evidence of cirrhosis of the liver, and invited comment on the diagnosis and further treatment.

DR. E. LEY said that he thought the condition was carcinomatous and that secondary deposits were present, and he advised a course of deep X ray therapy.

DR. J. B. COLQUHOUN said that he did not think there was sufficient evidence to exclude tuberculous disease of the spine which was not infrequently seen at that time of life. He thought he could exclude typhoid disease of the spine. Kyphos was present, with muscle spasm and pain on pressure over the ribs near the infected spine, which made him favour as a diagnosis tuberculosis rather than secondary carcinomatous deposits. In addition, the appearances in the films were suggestive of the presence of a tuberculous abscess with some calcification. He suggested that a Mantoux test should be performed, and if there was no response this result would be of value in the exclusion of tuberculous disease. If the lesion was tuberculous a sedimentation test under standardized conditions would indicate the degree of activity of infection. When the diagnosis was arrived at, Dr. Colquhoun thought that it might be justifiable to perform a fusion operation on the spine, which would at least make the patient lie down long enough to have a chance of recovery.

DR. C. H. OSBORN suggested that the injury might be traumatic; the patient had had a serious fall and a car accident. In the sedimentation test a rapid fall usually occurred both in actively infective and in malignant conditions; if this fall did not take place the result of the test would help to exclude both of these conditions. There was very little rarefaction to be seen in the films and not much evidence of inflammation; the symptoms and root pains were consistent, in his opinion, with a traumatic lesion.

DR. ERIC COOPER commented on the extraordinarily common association of cirrhosis and tuberculous lesions and agreed that the Mantoux and sedimentation tests should be carried out, though it was usual in cirrhosis to find a rapid fall in the sedimentation rate.

DR. C. J. O. BROWN thought that the fact that two bodies were involved favoured the diagnosis of tuberculous disease; neither trauma nor carcinoma was likely to affect two adjacent vertebral bodies. He did not think that a bone-graft operation would be suitable; he regarded the placing of a graft into a tuberculous spine as only a final fixative measure. At an earlier stage it would tend, if effective, to interfere with the natural process of collapse, scarring and healing. He was inclined to advise Dr. Andrew to continue the treatment that had been adopted and to add a spinal brace or jacket if necessary. The course of tuberculous disease at that age was either rapid or very chronic, Dr. Brown said, and he thought that he could detect in the film the shadow of a tuberculous abscess with some evidence of calcification; he thought it likely that with support of a minor nature the patient would continue to live for a number of years.

Dr. Andrew, in reply, said that he had decided to follow fairly closely the course of treatment that Dr. Brown had outlined. Drastic rest was difficult to impose on this

patient; he was the sole supporter of five childen and he was able to do his work, which consisted of attending to a machinery plant and did not involve any physical labour.

Rheumatic Heart Disease.

DR. R. D. SMITH showed two patients with rheumatic heart disease. One was a little girl, aged seven years, and the other a male patient, aged twenty-four years. The little girl had been healthy until she had had scarlet fever some eighteen months before the time of the meeting. She was admitted to hospital on September 16, 1937, looking very weak and ill. For a week she had had pain and slight swelling in the ankles, and she had been feverish and short of breath. The heart was slightly enlarged; there was a thumping impulse and a loud murmur in the apical area and the pulse rate was well over 120 beats per minute; the temperature was 38·6° C. (104·4° F.) just after her admission to hospital. After six days of salicylate treatment and complete rest the temperature subsided, but it had run up again and the pulse had increased. Dr. Smith said that the child was still very ill and was receiving sodium salicylate in a dose of 0·65 gramme (ten grains) every four hours; he would be happy to hear any suggestions for further treatment.

DR. A. P. DERHAM, in reference to the slight cyanotic appearance of the child, commented that in young children moderately early cyanosis was suggestive of the implantation of rheumatic infection on a previously existing congenital heart anomaly, though he was not sure that this was the case on the present occasion. He also stated that some years earlier he had been in the habit of advising the use of sodium salicylate in full dosage for a very long time, but more recently he had realized that this practice was fraught with danger and disadvantage to the patient. Even at the present dosage, if it was continued, the sodium salicylate might be the cause of poisoning or of acidosis; as an alternative he had found calcium aspirin useful with a smaller amount of sodium salicylate. In view of the storm that had arisen about the use of digitalis in toxic heart conditions, Dr. Derham thought that it might be of interest to state that he still thought that small doses of digitalis were at times helpful for the relief of tachycardia in rheumatic heart conditions; he had not seen it do harm except when extra-systoles were frequent.

DR. M. D. SILBERBERG stated that he could not see what function digitalis would serve in these conditions; at most it could be expected to increase the tonus of the heart muscle, but it would not do this in small dosage. He was doubtful if it was necessary, and he also doubted whether it would be successful in slowing the rate either with small or with large dosage in such a case.

DR. F. KINGSLEY NORRIS doubted the wisdom of using sodium salicylate at all; in the present instance the child had not been in pain on admission to hospital, and the infection had flared up while she was having the sodium salicylate. He considered that if the patient was in pain the salicylate could be used for twenty-four hours to relieve the pain, but that it should not be continued because of the anorexia or even more serious effects arising from it. Dr. Norris disapproved of the sedimentation test, and quoted the opinion of Dr. Rupert Willis that it was entirely useless and varied so much with the temperature of the room and the calibre or slant of the tube that any prearranged result could be obtained by attention to these factors.

DR. G. A. PENINGTON disagreed with what Dr. Norris had said about the value of the sedimentation test; under standardized conditions of temperature and calibre of the tube used, with the additional standardization of the erythrocyte count by dilution, it could be regarded in Dr. Penington's opinion as a valuable guide in indicating the progress from time to time in an individual case. He also referred to the importance of a high vitamin diet to allow an ample margin for deficient absorption; he emphasized the importance of raising the resistance to infection, of increasing absorption, and of building up the general condition of the patient.

DR. H. BOYD GRAHAM stated his conviction that prolonged convalescence and graduated and controlled restoration of activities, together with patient and prolonged after-care, were of the greatest importance in safeguarding to the utmost the future of a patient such as the little girl under discussion.

DR. S. WILLIAMS said that the salicylate preparations were all absorbed as salicylic acid, so it did not appear to matter very much which one was used to begin with. He also discussed briefly the question of vitamins, and stated that, according to reports of work done at Cambridge, there was a very definite deficiency in vitamin *C* in rheumatic fever and in pulmonary tuberculosis. He mentioned the advisability of the plentiful administration of orange juice or of a reliable synthetic preparation of ascorbic acid.

DR. J. BEGG raised the problem of the need for prolongation of treatment and for restriction of activity in the case of the young adult who had rheumatic pains insidiously over a period of months or years. Unless it was very certain that the restrictions would be beneficial, it was difficult to justify the infliction of hardship and the loss of earning capacity entailed.

DR. ERIC COOPER wondered whether the cyanotic tinge was caused by the salicylates; he advised discontinuance of this form of treatment in the case of the little girl shown. He also affirmed that the sedimentation test was only meant to be used as a basis for comparison of the results from time to time in any one case.

Dr. Smith, in reply, said that there was no doubt that the cyanosis was present before the patient's admission to hospital and before salicylate treatment was commenced. He was satisfied that prolonged complete rest was the main principle of treatment, and he was sorry that no one had expressed any opinion about the removal of the tonsils.

The other patient shown by Dr. Smith had been admitted to hospital on August 16, 1937. He had dislocated his shoulder on June 30, 1937, and subsequently had contracted a febrile illness which was regarded as gastric influenza and was followed by a consolidation of the base of the left lung, which had not cleared up until approximately August 9. An August 15 the right wrist and left knee became acutely inflamed; and later the left wrist, the right knee and both ankles were swollen and painful. On the patient's admission to hospital it was noted that the apex beat was well outside the nipple line, and, though the pulse was regular, a systolic murmur was audible over the apical area and was conducted out towards the axilla. Pulsation was visible and palpable over the second and third intercostal spaces on the left side and close to the sternum. The patient had some carious teeth and the throat was inflamed and reddened, but in other respects the findings were of a negative nature. Treatment was being carried out by means of rest in bed and full dosage of sodium salicylate at the time of the meeting.

DR. G. A. PENINGTON said that for patients intolerant of sodium salicylate he had found that sal ethyl carbonate was very well tolerated. It had been used in doses of 2·6 grammes (forty grains) every two hours for patients who could not tolerate twenty grains of sodium salicylate given every four hours. It should be generally realized that every patient with rheumatic fever had rheumatic carditis; within twenty-four hours of the onset he had been able to demonstrate pericardial friction sounds. Each patient in the acute phase should be regarded as the subject of acute carditis.

DR. C. H. FITTS said that he had been most interested to hear the expression of Dr. Penington's views, which, however, he thought were open to argument; at all events some of the patients recovered without apparent damage to the heart. He was not satisfied, for example, that the patient shown had heart disease; these cases did bring out, however, the problem of how to diagnose heart disease in rheumatic fever. Dr. Fitts agreed that rest was the main object of treatment, but in spite of prolonged rest mitral stenosis and aortic incompetence might develop.

DR. J. M. ANDREW said that the patient presented evidence of a cardiac lesion when he had seen him before admission to hospital; the apex beat was in the sixth interspace and well out towards the axilla, a systolic bruit was easily audible and galop rhythm was present.

DR. SILBERBERG expressed the opinion that the patient had toxic rheumatic myocarditis which would remain; he had the feeling that valvular disease would not develop; the virus of the infection was in the heart muscle and was slow in clearing up, as in other infectious illnesses.

Muscular Dystrophy.

DR. SMITH also showed a boy, aged thirteen years, who had been in hospital from July 24, 1936, to October 5, 1936, and again from November 5, 1936, to the time of the meeting. Four months prior to his first admission to hospital he had had what the mother thought was pneumonia, but no doctor had been consulted. The mother stated that before that illness he had been able to walk about and he had attended school, but he was in Grade III instead of Grade V; he had not walked since. When admitted to hospital on July 24, 1936, he had a fresh attack of pneumonia. Contractures of the ham-string tendons were present, and Dr. Trumpy, who had charge of his case, had tried to overcome the contracture unsuccessfully by means of weight extension, which had caused sloughing over the tendon behind the knees and of the flexors of the elbow. The mental condition was poor and many of the muscles were wasted. The tongue was large and flabby, some carious teeth were present, and the head looked relatively large. The patient had a silly, good-natured expression. The pupils were normal in size and in reaction to light. They had not been able to elicit the knee jerks or the superficial abdominal reflexes, but the ankle jerks were normally active and the plantar reactions were flexor in type. No disturbances of sensation had been demonstrable.

DR. A. P. DERHAM thought that the boy's case was a fairly typical example of pseudo-hypertrophic muscular dystrophy. The calf muscles were weak, the shoulder girdle was wasted, and atrophy of the small muscles of the hands was present; when asked to sit up, the boy had only been able to make a wriggling movement. Dr. Derham thought that a specimen of blood should be subjected to the Wassermann test and that further measures should be adopted to overcome the contractures of the knees.

DR. F. K. NORRIS wondered whether the patient was any more than a mentally deficient child who had developed secondary contractures from prolonged recumbency. He had found glycine treatment disappointing in the muscular dystrophies of childhood.

DR. J. B. COLQUHOUN said that he had not seen a patient with pseudo-hypertrophic muscular dystrophy who had reached at the age of thirteen years the advanced stage of the patient shown. He had noted that the boy's calf muscles contracted strongly, and in pseudo-hypertrophic muscular dystrophy in the advanced stage this power of contraction of the calf muscles would have been lost. It was difficult to supply a diagnosis without studying the case carefully, but Dr. Colquhoun would like the contractures to be wedged straight if this procedure proved feasible. It had occurred to him that it was possible that the solution of the diagnosis might be that the virus of poliomyelitis might have invaded the central nervous system of the boy and that the preexisting subnormal mentality had complicated the clinical picture.

DR. E. LEY said that while at Liverpool recently he had seen two patients with pseudo-hypertrophic muscular dystrophy who had developed worse contractures than those of the boy shown by Dr. Smith. He had also had the opportunity to try the effect of glycine treatment in doses of 15 grammes in the case of five boys in one family all affected by the condition; there had been considerable improvement in three of these boys, and there had been no subjective tremor of the muscles.

DR. ERIC COOPER recounted briefly the circumstances in which he had used glycine over a period of two years, at first in doses of 30 grammes a day and later in doses of 15 grammes a day, for a patient who had improved dramatically and was since able to dance and to milk cows; he thought it possible that at the age of eleven years the boy shown by Dr. Smith had had an acute illness which might have been encephalitis, the aftermath of which had been the development of the foot and face type of dyspituitarism.

DR. C. H. OSBORN thought that in view of the uncertainty of diagnosis it would not be unreasonable to postulate that the contractures were of a simple nature, the result of the lengthy period in bed. It was important to correct the deformity and to attempt to put the boy on his feet. A method he had used with satisfaction was to perform a muscle-slide operation of the muscles attached to the *tuber ischii* and to the surrounding bone.

DR. F. P. MORGAN said that he would like to support the diagnosis of muscular dystrophy, and he thought that it was unlikely to be Little's disease as had been suggested. He did not think that a muscle-slide operation and straightening of the limbs by this manœuvre would be likely to benefit the patient.

Dr. Smith, in reply, thanked those who had contributed to the discussion and emphasized the point that except for subnormal mentality the boy was well able to walk to school and was in apparent good health before the onset of the acute infection in March, 1936.

Buerger's Disease.

DR. T. A. F. HEALE, in the unavoidable absence of Dr. O. B. Goyen, showed a male patient, aged thirty-seven years, who was under Dr. Goyen's care at Warragul, but who had been seen by Dr. Heale at Saint Vincent's Hospital. In March, 1937, the man had had sudden severe pain in the second toe of the left foot which had become swollen and reddened. Thinking that the toe was infected, Dr. Goyen had removed the toenail, and the pain had become much worse, the lesion going on to gangrenous ulceration. Since then the third toe of the left foot had become affected and was still untreated. There was an absence of pulsation in the *dorsalis pedis* artery of the left foot, but pulsation was present in the other arteries. Dr. Heale said that the onset in this case was of interest and commented on the ease with which the condition could be in error diagnosed as an infection instead of a thrombotic condition. Another common difficulty encountered was to mistake the condition in the early stages for the effects of ingrowing toenail; if any operation was done the patient would promptly develop gangrene of the toe.

Carcinoma of the Right Humerus.

DR. C. M. LEY showed the other patient whom Dr. Goyen had intended to show. A man, aged sixty-one years, had come under Dr. Goyen's care on September 9, 1937, with a fluctuant mass over the right humerus which was evacuated through a small incision on September 11. A report on some biopsy material removed indicated that it came from an active malignant growth, probably a secondary deposit, and that possibly the primary growth was in the kidney.

Fracture about the Elbow Joint.

DR. D. I. FITZPATRICK showed a series of skiagrams to illustrate the results of an injury to a railway employee who had been thrown from a motor car. The patient had sustained a swelling around the elbow, and Dr. Fitzpatrick had opened the joint laterally. He thought he had removed all the pieces of bone, but a later skiagram showed that one was left. He had reopened the joint and removed the fragment with difficulty after probing. In a skiagram taken a month later a piece of bone still appeared unattached and in an abnormal position. The patient had recovered only ten degrees of movement with very little supination or pronation, but there was not any evidence of nerve lesions. Dr. Fitzpatrick asked for advice as to further treatment.

DR. C. J. O. BROWN thought that as the injury was three months old the arm should be left alone operatively, but that with active movements considerable improvement might be expected.

DR. J. B. COLQUHOUN considered that the case presented a very difficult problem. He recounted the details of some interesting examples of fractures about the elbow joint with which he had recently came in contact. In the case of a fracture about the elbow with a loose fragment complicated by dislocation, he had reduced the dislocation and waited a considerable time before removing the loose fragment; in the case of a fracture of the epicondyle he had not removed the fragment, but had reattached it, and in that way had obtained additional function. Dr. Colquhoun said that if operation for a good functional result was attempted while blood clot was still forming or present it was probable that the elbow would be stiff; it was better to wait till the effects of the trauma had settled down. He also expressed the opinion that the surgeon had left some periosteal tissue behind and that the new bone was appearing from it. He thought that later on, in the case of Dr. Fitzpatrick's patient, a bony arthrodesis in the optimal position should be carried out.

(To be continued.)

VICTORIAN BRANCH NEWS.

THE following items of news of interest to members of the Victorian Branch of the British Medical Association are published at the request of the Council of the Branch.

Medical Service under the Workers' Compensation Act.

In the Legislative Assembly on Tuesday, November 30, 1937, Mr. Field, M.L.A., asked the Honourable the Chief Secretary:

1. If it is a fact that the State Insurance Commissioner has sent a letter in the following terms to many policy-holders of the State Accident Insurance Office:

Sir,

Re Workers' Compensation Act.

I have to advise that I have appointed Dr..... of.....to represent this office in and around the.....District. In the event of any of your 'workers' meeting with an accident, I would ask you to be good enough—whenever necessary and possible—to arrange for them to see the doctor.

Now that the Amending Act makes provision for payment of medical fees, special arrangements have been made in various districts for doctors to give attention to disabled workers employed by policy-holders of this office, and this arrangement has so far proved most satisfactory in the interests of all concerned.

Yours faithfully,

(Signed) W. H. HOLMES,
Insurance Commissioner.

2. Whether the State Insurance Commissioner has any right under the *Workers' Compensation Acts* to interfere with and abrogate the long-established principle that a patient should have free choice of doctor.

3. If, in view of the possibility that the rights of an injured worker might be prejudiced by compelling him to attend for treatment a doctor employed by the Insurance Office, and thus depriving him of the services and advice of his family doctor, he will instruct the Commissioner to cancel and discontinue the practice of appointing doctors to treat injured workers coming under the provisions of the *Workers' Compensation Acts*.

The following answers were supplied:

1. Yes.

2. The employer insured has the right, under paragraph 13 of the Second Schedule of the *Workers' Compensation Act* and Part 4 of the Regulations thereunder, to arrange for the medical examination of a worker in receipt of compensation or of a worker who has given notice of accident.

Under the terms of the policy issued by the State Insurance Commissioner, all rights of the employer are conferred on the Insurance Commissioner.

3. It was not intended that the worker should be compelled to accept treatment by any particular doctor, this matter being at the discretion of the worker himself.

The main intention of the Insurance Commissioner, in the interests of the employer, the worker, and himself, was to obtain a medical report immediately an accident occurred.

The appointments were made by the Commissioner after due inquiry, and the local representatives of the State Accident Insurance Office were definitely advised that there was to be no interference in any way with other doctors. Furthermore, as a matter of professional etiquette, the doctors appointed by the Insurance Commissioner would not, under any conditions, interfere with the practices of fellow members of the British Medical Association.

To remove any ambiguity as to the position, the letter referred to will be redrafted to meet the position as above stated.

A Conviction under the Medical Act 1928.

On December 7, 1937, Fritz Koch appeared before the Rainbow Court on a charge that he, not being a person registered under the provisions of the *Medical Act* 1928 did pretend to be a doctor of medicine. The defendant did not appear, and was fined £50 with £2 1s. 8d. costs.

The Medical Act 1937.

The following circular has been received:

The Medical Board of Victoria desires to bring to the notice of medical practitioners that the above-mentioned Act (which is to be read and construed as one with the *Medical Act* 1928 and any Act amending the same) was assented to and came into operation on December 8, 1937.

Under the Act any registered practitioner who—

(*a*) demands payment from or renders an account to or collects fees from any person; or

(*b*) shares or agrees to share with any other practitioner any fee or other remuneration—

for or in respect of professional services rendered by any other practitioner, not being his partner under a partnership agreement approved and registered by the Medical Board, a permanent assistant in his sole employment, or a *locum tenens* in his practice, shall be liable for a first offence to a penalty of not more than £50, and for any subsequent offence to a penalty of not less than £50 nor more than £100.

It will be seen, therefore, that certain methods of rendering accounts and collecting fees for professional services which hitherto have been common practice are now illegal, and it is the desire of the Board that the least possible inconvenience will be caused to the public and the medical profession as a result.

The Board is of the opinion that—

1. Accounts for services rendered prior to December 8, 1937, will not be deemed to come within the provisions of the Act;
2. The expression "permanent assistant in the sole employment of a medical practitioner" means an assistant whose professional income is received entirely from the registered practitioner by whom he is permanently employed;
3. The term *locum tenens*, in addition to having the ordinarily accepted meaning, namely, a person acting for the time being during the temporary absence of a medical practitioner, will also include a substitute who is acting in an emergency on behalf of another practitioner;
4. The Act does not prevent a practitioner from informing a patient of the amount of the fee payable to a consultant, anæsthetist, pathologist, or assistant, and, at the time the service is rendered, requesting the patient to pay that fee to the consultant, anæsthetist, pathologist, or assistant (as the case may be).

It will be noticed that a partner, in order to be excepted from the necessity of rendering an individual account and from the provision that he shall not collect a fee or portion of a fee due to any other practitioner, must be practising under a partnership agreement which has been approved and registered by the Medical Board. Application for such registration, setting out the full name and professional address of each partner, accompanied by a statement in duplicate of the terms of the partnership agreement (one copy of which will be retained by the Board) and giving such other information as may in any particular case be required by the Board, should be forwarded in writing to the secretary to the Board without delay. Whenever a change is made or occurs in any of the particulars furnished in respect of any registered partnership agreement it will be the duty of each practitioner concerned to notify the Board thereof within fourteen days after such change.

Practitioners are requested to note carefully that a conviction under the Act will not only render the offender liable to a fine, but also may lead to the erasure or removal of his name from the Medical Register under the provisions of the *Medical Act* 1933.

A. S. JOSKE, J.P., M.D.,
President.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Leleu, Charles John Newhill, M.B., B.S., 1936 (Univ. Sydney), Kembla Street, Wollongong.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Apps, Ray Clifton, M.B., B.S., 1935 (Univ. Sydney), Newcastle Hospital, Newcastle.

Barclay, Francis Edison, M.B., 1924 (Univ. Sydney), 10, Bondi Road, Waverley.

Bowen-Thomas, Glynn, M.B., B.S., 1935 (Univ. Sydney), c.o. Bank of New South Wales, 47, Berkeley Square, London.

Harrison, Keith Selwyn, M.B., B.S., 1936 (Univ. Sydney), 153, Anzac Parade, Kensington.

Langton, Colin William Chisholm, M.B., B.S., 1937 (Univ. Sydney), 199, Hopetoun Avenue, Vaucluse.

Speight, Patrick Howard, M.B., B.S., 1937 (Univ. Sydney), 15, Mandalong Road, Mosman.

Obituary.

ALFRED WALTER CAMPBELL.

WE are indebted to Dr. L. R. Parker for the following account of the career of the late Dr. Alfred Walter Campbell:

In the death of Dr. A. W. Campbell, of Macquarie Street, Sydney, on November 4, 1937, the medical profession of Sydney has lost one of its most eminent personalities. His loss, however, is far from having a merely local significance, for his reputation as a neurologist, alienist, pathologist, research worker, writer and teacher was confined not to his own city and continent alone, but extended throughout the whole medical world. The results of his original research and investigation have been delivered before various of the most prominent of the British and continental medical and scientific societies, or have appeared in the most classical journals. His monumental book on "The Localization of the Cerebral Function" (self-illustrated with consummate draughtsmanship) was published by the Royal Society of London in 1905, and is still the accepted authority on that intricate subject. A thorough knowledge of French, German and Italian, moreover, permitted him to contribute original matter to the journals and societies of those countries, and was also responsible for his appointment as reviewer of foreign publications to many of the most famous British journals.

During the period of his most intensive work in England and on the Continent he was the associate and colleague of many men whose names are household words throughout the medical world—among them Sir James Crichton Browne, Byrom Bramwell, Professor Krafft-Ebing and Professor Rutherford. The personal tributes of these and many other eminent men to Dr. Campbell's attainments and achievements are most arresting. Crichton Browne stated that A. W. Campbell had a most distinguished place amongst the pathologists of the day; and McDowall, of Northumberland County Asylum, wrote on April 8, 1904, that he was the most distinguished scientific worker connected with the English asylums. Regarding his book, Byrom Bramwell wrote, "It is a most magnificent piece of work and will be recognized all over the world"; while W. Bevan Lewis wrote, "It is a magnificent piece of work, and no asylum library in particular can afford to be without a copy". A comparatively recent tribute to Dr. Campbell's eminence was his appointment as vice-president to the section of neurology at the centenary meeting of the British Medical Association in 1932.

Alfred Walter Campbell was born on January 18, 1868, on his father's station at Cunningham Plains, near Harden. He was educated at Oakland School, near Mittagong, the headmaster—whom he intensely admired—being a Mr. Southey, son of the English poet. In 1885 he entered the Medical School of the University of Edinburgh, from which he graduated as M.B., Ch.M. with honours in 1889. He at once decided to devote his life to research in mental science, and accordingly took successive assistantships in several British mental hospitals. To further his experience, he then proceeded to Vienna, where he was associated with various famous continental professors, including Krafft-Ebing, to whom he was assistant for a considerable period. Later he went on to Prague, where he was appointed to the staff of the State asylum in that city.

On his return to England in 1892 his thesis on "The Pathology of Alcoholic Insanity" secured for him the degree of Doctor of Medicine at the Edinburgh University, and, being highly commended by the Senate, he was accorded the honour of receiving a gold medal. In this year he was appointed by the Lancashire County Asylums Board to the dual positions of resident medical officer and director of the pathological laboratory at the Rainhill Asylum, Liverpool, one of the largest and most important of its type in England. At this stage the laboratory was in its modest infancy, but under Dr. Campbell it was entirely rebuilt and equipped with all modern pathological and bacteriological apparatus, was affiliated as a teaching school with the University of Liverpool, and became a place of visitation and study by specialists from all parts of the world.

Having spent twenty intensive years abroad and wishing to return to Australia, he, in 1904, made application for the vacant position of Inspector-General of Insane in Victoria. In view of his impressive record, his Australian birth, and an unprecedented series of recommendations from almost every eminent neurologist in England, it seems impossible that any other candidate could have outweighed his claims; but it was characteristic of the man that, hearing that a local applicant, whose work he admired, was in the field against him, he promptly withdrew his candidature.

Returning to Australia in 1905, he entered upon private practice in Sydney as a specialist in neurology and mental diseases. Eminence was already his, and he practised with distinction from then on to the date of his retirement early in 1937. During this period he continued to follow his bent for research, and he often enough said that had it been merely a matter of choice and opportunity, he would never have abandoned the scientific for the practical side of his profession.

In 1906 he married Miss Jenny Mackay, daughter of David Mackay, of Wallen Been, a station adjoining that on which Dr. Campbell himself had been born. These two had been friends since childhood, and her brother, of Central Australian fame, formed the third in a very united

coterie. His two daughters, Veda and Helen, were born in 1907 and 1909 respectively.

In 1914 Dr. Campbell was one of the first to volunteer for war service and was sent to Egypt. At the expiration of his term there he endeavoured to secure a position in the European zone, but this not eventuating, he was subsequently appointed to the Military Hospital at Randwick, which appointment he continued to hold up to the time of his death. His other important honorary positions were those of Consultant Neurologist to the Royal Alexandra Hospital for Children, the Coast Hospital, and the Department of Repatriation.

With his fine strength and physique it is easy to conceive that he would excel in athletic exercises. He played golf, but he loved cricket as the "best of games", with football a close rival. He had the signal honour of being captain of both cricket and football teams of the Edinburgh University. Happiest among his reminiscences were his experiences as a member of the Edinburgh-Australian Cricket Club, whose practice was to make picnic tours during vacations through England and Scotland. On one of these he was associated with Judge Cecil White and Dr. "Roley" Pope, and had for an opponent the celebrated and rather truculent Dr. E. M. Grace. He was always more reticent about his medical than his sporting achievements, so it was quite characteristic that when asked (with professional significance) what he did during his English period, he unexpectedly said that when he wasn't playing cricket he was shooting grouse.

In later years, as activities in sport diminished, his leisured interest became increasingly centred in his home and its surroundings. The house, set high on the steep hillside, approached by a winding hairpin-bended drive, and hidden by trees through which an occasional flash of the loch-like waters of the bay could be seen, always seemed peculiarly suited to his reserved temperament and Scottish extraction. Here, although intensely interested in the flowers and trees, he was even more so in the numerous and varied birds which made them their sanctuary. He knew all their names and their habits, and he perseveringly diverted his grouse-shooting abilities to the task of protecting them from their hereditary enemies. Inside the house multitudes of books and prints demonstrated his interests, which in these matters were very considerably historical. In this regard students of romantic history may not be uninterested to know that he did not approve of the implacable attitude of his ancestor of Argyll towards tragic Mary Queen of Scots.

Despite his characteristic and Caledonian reserve, his humanity made him not hard to know. He was an excellent companion. His knowledge and experience were profound, his reading wide and his sense of humour keen, non-hilarious and pleasantly sardonic. His smile, always attractive, with a gleam from under the deep brows, and the corners of the closed lips slightly drooped, was a photographic image of his humour. I do not know whether he was a better listener or talker. As a listener he had a most attractive habit of definitely expressing approbation if the subject or manner of telling pleased him. "A good story and well told", he would say. His own speech was delightful, his enunciation infinitesimally studied, but not over-emphasized, the choice of words excellent, the vocabulary unplumbed, and the clear-headed, onward, unhurried flow something for a more halting companion to both envy and admire. Possibly he cultivated the art of speech because of his frequent necessity for addressing large medical or scientific gatherings; but it was typical of him to do all things well—to spare no pains. Unpleasant voices perturbed him strangely, and I can believe the story that he once invoked the personal aid of a colleague whom he barely knew, not because of any peculiar medical virtue, but because he liked the way he had once "said something".

His sincerity regarding anything associated with his work was rigid. Once asked whether he would like to have been a teacher, his emphatic "No" was startling. "It would have taken too much out of me", he added, "the constant dread that I should leave something out, or say something which might not be true."

His knowledge of his own intricate specialty was profound, and his mental clarity regarding it fluent and inextinguishable. Even in the fell clutch of his material ill, with his brain blurred by the partial anodynes, his unconquerable mind traversed the intricate tracts and terminology of his own special region, and translated the march of the enemy in a fashion that was terrifying; but his courage was equal and parallel to his knowledge.

His humour was no less ingrained and brave. Almost his last conscious speech was a story, and although it was only with tense determination that he clung to its misty and elusive thread, still he pressed on with every right word in perfect sequence and made his final and triumphant point.

When one has stood by the side of a colleague and friend in a long journey through the valley of the shadow, the desire to say what might be thought too much is almost irresistible. On the other hand, to withhold it seems to deny an honour that is justly due. Whatever may be the correct and proper mean, if what is set down herein serves to show that Alfred Campbell lived as fully as few may hope to live, and died as heroically as any might hope to die, then it tells but a simple truth.

It may well then be taken for granted that the medical profession of this city and State in particular is well aware that it has lost one of its most distinguished members: one who was esteemed for his culture and attainments, honoured for his high character and achievements.

and held in most affectionate regard by those privileged to rank among his personal friends. The roll of those names that stand high in the medical world of Australia is a steadily growing one; but in that, in the intensive search for truth, he blazed a new and intricate trail, and thereby added to the sum of human knowledge, the name of A. W. Campbell must always stand amongst the very highest of them all.

Professor W. S. Dawson writes:

It has been said that great men bequeath to posterity the task of explaining them. The task is sometimes an inspiration and a privilege.

The name of A. W. Campbell will endure for his great work on the histology of the cerebral cortex, which forms a definite landmark in the study of the brain. After some years of general and special experience in pathology, including a visit to the leading neurological laboratories on the Continent of Europe, where doubtless Germanic thoroughness and industry appealed to his orderly mind, Dr. Campbell set himself to his great work, the significance of which stands out upon a consideration of contemporary knowledge concerning the cerebral cortex.

Prior to 1870 the view prevailed that the cortex functioned as a whole, and that lesions produced a general depression of activity in proportion to their severity and extent. It is true that phrenologists had described and "localized" some twenty-seven different faculties, while in 1861 Broca had published an article in which he provided clinical and pathological evidence in favour of placing speech in the third left frontal convolution. Then in 1870 and later, Hughlings Jackson published his clinical and pathological studies on fits, and in 1873 described "Jacksonian" epilepsy, with its march of movements initiated by lesions in certain regions of the brain. In 1870 also Hitzig and Fritsch had shown experimentally that the cortex is not only excitable, but that excitation at certain definite points is followed by movements on the opposite side of the body. Further, they showed that destruction of these excitable areas results in paralysis of those movements which had previously been evoked. During 1873 to 1876 Ferrier repeated these experiments. In 1872 Meynert published his studies on the lamination of nerve cells in the cortex and described intergyral association fibres. In 1874 Betz described the large pyramidal cells which bear his name, and in 1878 Bevan Lewis showed that the area responsible for the initiation of voluntary movement contains cells which are distinctive in their size and arrangement. A year later Bevan Lewis and Henry Clarke defined this motor area more accurately, and were thus the first to correlate exactly functional and histological areas. Between 1877 and 1890 H. Munk published several papers in which he described a number of sensory spheres in the Rolandic area, and suggested that sensory paralysis must precede paralysis of movement. Schäfer and others later provided conclusive experimental and clinical evidence that paralysis of movement could not result from loss of tactile sensibility alone.

To return to histology, Theodor Kaes during 1891 and following years made a survey of medullated fibres throughout the cerebral cortex, using a modification of Weigert's hæmatoxylin stain. Vulpius in 1892 published a description of tangential and association fibres. Then between 1898 and 1903 Flechsig contributed his studies of myelination and described some forty fields grouped into the primordial, myelinated before birth, and the intermediate and terminal, myelinated within a month of, and more than a month after, birth. In 1895 Carl Hammerberg contributed to the description of cell lamination, but did not attempt to make a complete survey of the cortex. In 1900 to 1903 Ramón y. Cajal made a comprehensive study of motor auditory, visual and olfactory areas based on the histological appearances revealed by the staining methods of Nissl, Weigert and more especially of Golgi. But while Cajal differentiated various areas according to their distinctive types of cells, he did not attempt a determination of their boundaries. In 1901 Sherrington and Grünbaum published their physiological studies on the cerebral cortex in the higher apes. The brains of several of these experimental animals were subsequently examined by Campbell.

It was at this stage that Campbell decided to piece together what he very aptly termed "our disjointed knowledge of the structure of the cerebral cortex". Campbell's outstanding contribution to science is his accurate, carefully oriented, minute and comprehensive survey of the structure of the whole of the human *cortex cerebri*. Some idea of the prodigious labour involved may be gained from the fact that he examined some fifty blocks from each of twenty-five brains, each block being cut at about 100 levels, sections from each of which were stained for fibres and cells. Several cerebral hemispheres were cut throughout into serial sections stained alternately for cells and fibres. The results of this research were published in 1905 in "Histological Studies on the Localization of Cerebral Function", part of the cost of which was met by the Royal Society of London, to which the work had been submitted in 1903. The monograph contains numerous diagrams from Campbell's hand, and drawings by a colleague at Rainhill Asylum, Dr. A. C. Wilson. Several of these illustrations are still being reproduced in textbooks on anatomy and neurology. In defining the motor area, Campbell included in his material brains from cases of amyotrophic lateral sclerosis and from patients who had some considerable time prior to death undergone amputation of limbs, and was able to show that atrophy in the motor region was restricted to Betz's cells, that is, to the precentral convolution. In the sensory field, Campbell used brains from cases of *tabes dorsalis*. While he regarded microscopic study of the motor field as complementary to the work of the physiologist and clinician, he declared that "histology alone can give the precise extent and limits of sensory stations". Altogether some dozen areas were defined: the precentral and intermediate precentral (motor) area, the postcentral and intermediate postcentral (sensory) areas, the occipital and intermediate occipital, temporal and intermediate temporal, parietal, frontal and prefrontal, the insula (Reil) and limbic or olfactory areas. Such was the work which Tilney and Riley term "pre-eminent as one of the most illuminating of all efforts towards more correct cerebral localization". The composite diagrams of Campbell's and Brodmann's (1909) areas no doubt continue to be familiar to every student of physiology.

Little of importance has been changed in Campbell's descriptions or added to them by subsequent investigators. An addendum to the monograph contains some comparative studies of the brains of the pig, cat and dog.

The phylogenetic studies of Elliot Smith and others have provided a clue to much that Campbell found obscure in his own work. Speaking of the frontal and prefrontal areas, he said: "The portions which I have just defined comprise a territory, the functions of which are too little known. It is a part where theory holds the major hand." Something of value has come out of comparative anatomical studies of this region, while the crude experiments of war and the finer technique of the surgeon have provided informative material for the physiologist and the medical psychologist.

Amongst the numerous papers which Campbell published while pathologist to the Lancashire County Asylums Board, two call for special notice: "The Pathology of Herpes Zoster" (1900, in association with Henry Head), in which thoracic and other sensory zones were defined by correlating clinical appearances with pathological changes in the spinal root ganglia and tracts, and a paper on "Tracts of the Spinal Cord and Their Degeneration", which appeared in 1897. This was a critical digest derived from an extensive survey of the literature which served for years as a reliable work of reference. In 1919 to 1920, in conjunction with Professor Cleland, Campbell published several papers on acute encephalomyelitis (the Australian "X" disease), which was satisfactorily proved to be due to a virus transmissible to certain animals as well as to man. On numerous other subjects relating to neurology and psychiatry Campbell wrote and spoke with a precision,

logic and carefully chosen phraseology which added as much elegance as sound argument to discussions.

In 1935, on the occasion of the centenary of the birth of Hughlings Jackson, he delivered an oration to the Section of Medical History (British Medical Association) in Sydney without reference to notes, which will remain in the memory of his audience as a most worthy tribute to the great teacher whose rounds he had attended at Queen Square.

When finally "Nature bore him to the door of death", it is pleasant to recall that he could turn his mind from his painful illness to happy episodes of his earlier life, and that the pride of past achievements provided some compensation for the termination of an active life.

Dr. J. A. L. Wallace writes:

As former Secretary of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association, I should like to add a tribute to the memory of the late Dr. A. W. Campbell.

The Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association was formed in 1924, and Dr. A. W. Campbell, the then president of the same section in the first session of the Australasian Medical Congress (British Medical Association), was one of the six convenors of the inaugural meeting held on June 5, 1924, the others being Dr. Andrew Davidson (President of the New South Wales Branch of the British Medical Association), Dr. Eric Sinclair (Inspector-General of Mental Hospitals), Sir John McPherson (Professor of Psychiatry, University of Sydney), Dr. A. E. Mills (Dean of the Faculty of Medicine, University of Sydney), and Dr. John I. Hunter (Challis Professor of Anatomy, University of Sydney).

Following Sir John McPherson, who was the first chairman of the section, Dr. A. W. Campbell held this office in 1925, and his genial and impressive personality was a powerful influence in holding the interest of the members of the section.

During the first year of the section, the first ordinary meeting was held at the A.J.C. Home for Soldiers and Sailors, Canonbury, mainly by the activities of Dr. Campbell, who demonstrated that afternoon in a most interesting manner several neurological cases.

Throughout the following years the one member of the committee who could almost invariably be relied upon to be present at meetings was Dr. Campbell, and his well considered views for the arrangement of the various subjects of the section and contributions for meetings and to discussions were invaluable. He again held office as chairman in 1932, and on that occasion his retiring address was "Reminiscences of Hughlings Jackson", which was given in his usual deliberate, clear and concise style, and was delightful to listen to.

Every member of the section, in common with all who knew the late Dr. A. W. Campbell, will realize the serious loss his death has been to the specialty.

The following is a list of the late Alfred Walter Campbell's contributions to scientific publications and organizations:

"*Ein Beitrag zur pathologischen Anatomie der sogenannten Polyneuritis Alcoholica*", *Prager Zeitschrift für Heilkunde*, 1892.

"A Further Contribution to the Morbid Anatomy of Polyneuritis Alcoholica", *Liverpool Medico-Chirurgical Journal*, 1893.

"A Contribution to the Morbid Histology and Pathology of the Neuro-Muscular Changes in General Paralysis of the Insane", *Journal of Mental Science*, 1893. (An essay which gained the medal and prize offered by the Medico-Psychological Association.)

"On Vacuolation of the Nerve Cell of the Cerebral Cortex", *Journal of Pathology*, 1894.

"A Case of Thrombosis of the Inferior Cerebellar Artery with Cord Lesion", *Liverpool Medico-Chirurgical Journal*, 1894.

"On Degenerations Consequent on Destructive Lesions of the Human Cerebellum", *The British Medical Journal*, 1894.

"The Morbid Changes in the Cerebro-Spinal Nervous System of the Aged Insane", *Journal of Mental Science*, 1894.

"The Breaking Strain of the Ribs of the Insane. An Analysis of a Series of Cases Tested with a Specially-Devised Instrument." (Paper read before the Medico-Psychological Association, London, 1894.)

"The Breaking-Strain of the Ribs of the Sane compared with that of the Insane." A sequel to the last-mentioned investigation. (Paper read before the Medico-Psychological Association, London, 1895.)

"The Histology and Pathology of Bone Degeneration." (Paper read before the Liverpool Medical Society, 1895.)

"Sensory Aphasia in the Insane." (Paper read before the Liverpool Medical Society, 1894.)

"On the Uses of Formic Aldehyde in the Pathological Laboratory." (Communicated to the Medico-Psychological Association, Newcastle, 1895.)

"On the Course of the Pyramidal Tracts." (Communicated to the Medico-Psychological Association, Newcastle, 1895.)

"Heterotopia Spinalis." (Communicated to the Liverpool Medical Society, 1896.)

"The Pathology of Acute Pancreatitis." (Paper read before the Liverpool Medical Society, 1896.)

"Colitis or Asylum Dysentery: An Anatomical, Bacteriological and Experimental Research", *Journal of Pathology*, 1896.

"The Etiology of Asylum Dysentery, So-Called." (Communicated to the Medico-Psychological Association, London, 1897.)

"On the Tracts of the Spinal Cord and their Degenerations: A Critical Digest", *Brain*, 1897.

"The Pathology of Disseminated Sclerosis." (Communicated to the Liverpool Medical Society, 1897.)

"The Relationship between Syphilis and General Paralysis of the Insane." (Opening a discussion at a meeting of the British Medical Association, Portsmouth, 1898.)

A demonstration on various diseases and degenerations of the spinal cord before a meeting of the British Medical Association, Edinburgh, 1897.

"Points in the Normal Anatomy of the Pineal Gland." (Communicated to the Liverpool Medical Society, 1899.)

"On Certain Pathological Changes in the Pineal Gland." (Communicated to the Pathological Society of London, 1900.)

"The Changes in the Nervous System in Cases of Pernicious Anæmia." (Communicated to the Liverpool Medical Society, 1899.)

"The Histology of Cerebral Tumours." (Communicated to the Liverpool Medical Society, 1894.)

"On Amyotrophic Lateral Sclerosis." (Communicated to the Liverpool Medical Society, 1899.)

"The Arrangement of Nerve Cells and Nerve Fibres in the Cortex Cerebri in Cases of Idiocy." (Communicated to the Medico-Psychological Association, London, 1900.)

"The Pathology of Herpes Zoster and its Bearings on Sensory Localization" (in collaboration with Henry Head, F.R.S.), *Brain*, 1900.

"The Frequency of Herpes Zoster in the Insane." (Communicated to the Liverpool Medical Society, 1900.)

"Changes in the Vesicular Column of Clarke in Cases of Descending Degeneration of the Pyramidal Tracts." (Communicated to the Neurological Section of the British Medical Association, 1897.)

"The Distribution of Certain Fibres in the Posterior Columns of the Cervical Cord, with Remarks on Asystemic Posterior Column Scleroses." (Communicated to the Neurological Section of the British Medical Association, 1897.)

"On Changes in the Spinal Cord in consequence of Amputations." (Communicated to the Liverpool Medical Society, 1897.)

A demonstration on alterations of cortical lamination in cases of idiocy before the International Medical Congress, Paris, 1900.

"The Medullated Nerve Fibres of the Cerebral Cortex", *Liverpool Medico-Chirurgical Journal*, 1902.

A demonstration on the arrangement of nerve fibres in the normal *cortex cerebri* before the annual meeting of the Medico-Psychological Association, 1902. An extension of the last demonstration before the annual meetings of the British and British Medical Associations, 1902.

"On Auditory Localization and Insane Hallucinations." (Communicated to the Otological Society, 1903.)

"A Study of the Brain in Cases of Amyotrophic Lateral Sclerosis and Amputation as a Guide to Motor Localization." (Communicated to the Liverpool Medical Society, 1902.)

"The Histology of the Motor Area in the Anthropoid Ape" (an addendum to the paper by Professor Sherrington and Dr. Grünbaum on the motor area), *Proceedings of the Royal Society of London: Philosophical Transactions*, 1904.

"Research on the Arrangement of Nerve Cells and Nerve Fibres in the Cortex Cerebri in Conditions of Health and Disease." (Communicated to the Anatomical Society of Great Britain, 1903.)

A demonstration on the last subject before the Medico-Psychological Association at Morpeth in 1903.

"Histological Studies on Cerebral Localization", *Proceedings of the Royal Society of London*, 1903.

"On the Localization of Cerebral Function." (Communication to the Neurological Society, London, 1904.)

The following is a list of the papers published by Dr. Campbell after 1904:

"The Homologies of the Rolandic Region", 1905; "Further Histological Studies on the Localization of Cerebral Function: The Brain of Felis Canis compared with that of Homo", 1904-1905; "Histological Studies of the Localization of Cerebral Function", 1905 (a monograph published with the aid of a grant from the Royal Society); "Cerebral Sclerosis", 1905; "On the Localization of Cerebral Function", 1907; "A Case of Syringomyelia", 1913; "Remarks on Some Neuroses and Psychoses in War", 1916; "A Contribution to the Experimental Pathology of Acute Poliomyelitis (Infantile Paralysis)", with J. B. Cleland and B. Bradley, 1916; "A Clinical and Anatomical Report of a Case of Friedreich's Disease", with W. F. Litchfield and O. Latham, 1917; "A Contribution to the Experimental Pathology of Acute Poliomyelitis (Infantile Paralysis)", with J. B. Cleland and B. Bradley, 1918; "The Nature of the Recent Australian Epidemics of Acute Encephalomyelitis: Successful Conveyance of the Virus to a Sheep, a Calf and a Horse", with J. B. Cleland, 1919; "Acute Encephalomyelitis: A Clinical and Experimental Investigation of an Australian Epidemic", with J. B. Cleland, 1919; "An Experimental Investigation of an Australian Epidemic of Acute Encephalomyelitis", with J. B. Cleland, 1919; "A Case for Diagnosis (Thomsen's Disease)", 1919; "The Epidemiology of Acute Encephalomyelitis (X Disease) in Australia", with J. B. Cleland, 1919-1920; "The Australian Epidemic of Acute Encephalomyelitis: A Consideration of the Lesion", 1920; "A Case of Nervous or Hysterical Fever", with L. Dowling, 1920; "Epilepsies of Childhood", 1927; "Cerebro-Spinal Syphilis", 1930; "Affections of Peripheral Nerves", 1931; "Treatment of Migraine", 1933; "The Nervous Child", 1933; "Dr. John Hughlings Jackson", 1935.

Correspondence.

A CORRECTION.

SIR: In the annual report of the Council of the Queensland Branch, published in THE MEDICAL JOURNAL OF AUSTRALIA of January 1, it is stated that Dr. Eustace Russell is believed to be the first Australian member to be elected a Fellow of the Royal College of Physicians of Edinburgh. This supposition is incorrect. The late Dr. Hugh Lathrop Murray, of Melbourne, was elected to this Fellowship in 1904; he was an Australian member until his death in 1929.

Yours, etc.,

Maribyrnong, W.3, Victoria,
January 12, 1938.

H. M. L. MURRAY.

THE TREATMENT OF TRACHOMA.

SIR: I have just read the masterly article on trachoma in the second volume of Duke Elder's text-book of ophthalmology, recently published. In the section devoted to treatment a note of extreme pessimism is sounded: "Nevertheless, no specific is known; and the only safe thing to say is that, while by destroying the diseased tissue with much pain and tribulation, trachoma may be rendered inactive, it is *incurable*". (Italics are mine.) Referring to daily treatment with "blue stone", which he says is still the most widely approved method, he proceeds: "It is a fairly true generalisation to say that every case treated thus from the beginning eventually becomes inactive, although *the process may take several years*." (Italics are mine.)

Discussion with colleagues in this and other States has convinced me that the quotations given express the opinions of the majority of ophthalmologists.

For many years I have encouraged a much more hopeful outlook. I agree that, if by cure "*restitutio ad integrum*" is understood, cure can be hoped for only in cases treated very early and before pannus or deep-seated infiltration of the tissues of the lids has taken place. I affirm, however, that the stage of "inactivity" may be reached and continue permanent in the great majority of cases, whether recent or old, with certainty and in a relatively short space of time. Like the chronic ulcer of the out-patient department, cases of trachoma are in danger of being neglected unless they are taken in hand in a spirit of confidence. They pass from hand to hand and encumber the clinic for years, to the weariness of patient and attendants alike. This is quite unnecessary. Adequate treatment can quickly clear up these chronic cases and restore the sufferers to the ranks of efficient members of the community. I have no specific to offer or any novelty to recommend. The essentials of treatment are threefold:

(1) The complete expression of all soft trachomatous material both from the lower as well as the upper lids. For this I work thoroughly with Graddy's forceps under a general anæsthetic and follow at once by painting the abraded lid surfaces with a solution of *Hydrargyri Perchloridum* 4% in glycerine.

(2) Painting the conjunctiva of both lids with this solution three times a week, care being taken to protect the cornea by dextrous use of swabs and fingers.

(3) And of first importance, the personal conduct of the case from start to finish by the surgeon himself. All delegation of the duty to colleagues fatally divides the responsibility and militates against the success of the treatment.

By these methods I will undertake to clear up any trachoma clinic and induce smooth cicatrization in three months.

Yours, etc.,

2, Collins Street, Melbourne, C.1,
January 12, 1938.

EDWARD L. GAULT.

THE MEDICAL BENEVOLENT ASSOCIATION OF NEW SOUTH WALES.

SIR: The Council of the Medical Benevolent Association of New South Wales wishes to convey its thanks to all those who so generously contributed to the Christmas appeal, issued in December.

The total sum collected amounted to £192 4s., of which £115 was immediately distributed to twelve deserving members, and the balance was placed to the credit of the general fund.

Yours, etc.,

J. M. GILL,
Honorary Secretary.

135, Macquarie Street, Sydney,
January 13, 1938.

Congresses.

INTERNATIONAL CONGRESS OF DERMATOLOGY AND SYPHILOLOGY.

THE tenth International Congress of Dermatology and Syphilology will be held in New York City, United States of America, in September, 1940. Dr. Oliver S. Ormsby is President, and Dr. Paul A. O'Leary, Mayo Clinic, Rochester, Minnesota, United States of America, is Executive Secretary.

Books Received.

A MANUAL OF TUBERCULOSIS FOR NURSES AND PUBLIC HEALTH WORKERS, by W. A. Underwood, M.A., B.Sc., M.D., D.P.H., with an introduction by J. R. Currie, M.A., M.D., F.R.C.P., D.P.H.; Second Edition, revised and enlarged; 1937. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 433, with illustrations. Price: 8s. 6d. net.

Diary for the Month.

JAN. 26.—Victorian Branch, B.M.A.: Council.
JAN. 28.—Queensland Branch, B.M.A.: Council.
FEB. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
FEB. 2.—Western Australian Branch, B.M.A.: Council.
FEB. 2.—Victorian Branch, B.M.A.: Branch.
FEB. 3.—South Australian Branch, B.M.A.: Council.
FEB. 4.—South Australian Branch, B.M.A.: Branch.
FEB. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
FEB. 15.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
FEB. 23.—Victorian Branch, B.M.A.: Council.

Medical Appointments.

Dr. T. W. Burgess has been appointed Government Medical Officer at Denman, New South Wales.

* * *

Dr. R. A. Robertson has been appointed Government Medical Officer at Albury, New South Wales.

* * *

Dr. C. H. Horsley has been appointed Government Medical Officer at Bourke, New South Wales.

* * *

Dr. T. H. R. Mathewson has been appointed Acting Director of the Child Welfare Department, pursuant to the provisions of *The Maternity Act*, 1922, of Queensland.

* * *

Dr. S. V. Marshall has been appointed Honorary Anæsthetist to the Randwick Auxiliary Hospital, Randwick, New South Wales.

* * *

Dr. A. W. Bowman and Sir H. Gengoult Smith have been appointed Certifying Medical Practitioners and Medical Referees at Melbourne, Victoria, pursuant to the provisions of the *Workers' Compensation Acts*.

* * *

Dr. K. Moss has been appointed a Visitor to the Heathcote Reception Home, in accordance with the provisions of the *Mental Treatment Act*, 1927, of Western Australia.

* * *

Dr. M. Schneider has been appointed Honorary Consulting Ophthalmologist, and Dr. C. Gurner Honorary Consulting Radiologist at the Morris Hospital and the Bedford Park Sanatorium, South Australia.

* * *

The following appointments have been made at the Adelaide Hospital, Adelaide, South Australia: Dr. G. A. Lendon, Honorary Physician; Dr. E. F. Gartrell, Honorary Assistant Physician; Dr. D. K. McKenzie, Clinical Assistant to the Medical Section; Dr. R. L. Verco and Dr. B. E. Wurm, Honorary Clinical Assistants to the Gynæcological Section.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," pages xvi to xix.

COMMONWEALTH OF AUSTRALIA, DEPARTMENT OF HEALTH: Temporary Medical Officer.
COMMONWEALTH OF AUSTRALIA, REPATRIATION COMMISSION: Consultant Neurologist.
PUBLIC SERVICE COMMISSIONER, ADELAIDE, SOUTH AUSTRALIA: Resident Medical Officer.
ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.
TARA DISTRICT HOSPITAL, TARA, QUEENSLAND: Medical Officer.
TOOWOOMBA HOSPITALS BOARD, TOOWOOMBA, QUEENSLAND: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135 Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178 North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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